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The Journal of

Thoracic Surgery

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VOL. 36, JUNE-DECEMBER, 1958
THE C. V. MOSBY COMPANY 1958 ST. LOUIS

VOLUME 36

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The Journal of Thoracic Surgery

VOLUME 36
JULY 1958
NUMBER 1

Original Communications

HIGH DEFECTS OF THE ATRIAL SEPTUM

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INTRODUCTION

With the extension of surgery to defects within the heart these defects have quickly become the subject of intense study. New information concerning many of them has appeared and there has been a wider dissemination of old knowledge. Attention has been paid to the abnormal anatomy and to the abnormal function and new diagnostic techniques have been developed. Special problems and unusual complications which may be associated with the surgical treatment of these defects have been described. In this presentation, I will deal with some of these features of one particular defect—a subtype of the atrial septal defects. The complications of surgical repair will be emphasized.

PATHOLOGIC ANATOMY

Foramen Ovale Defect.—The most frequent anatomic type of atrial septal defect is the foramen ovale defect (Fig. 1, *A*). Among 66 patients with atrial septal defects, 78 per cent were of this type. Fortunately, this is the easiest type to repair. There are no dangerous structures near it and there is enough septal wall around it so that the edges may simply be sewn together. It has been repaired successfully by either closed or open operative techniques, although I favor an open technique, employing hypothermia.

Low Defect.—The defect illustrated in Fig. 1, *B*, and designated as a low, a persistent ostium primum, or an incomplete type of common atrioventricular canal defect presents a much more serious problem. One edge of the defect is

From The Department of Surgery, Northwestern University Medical School, Chicago 11, Ill.

Received for publication Dec. 23, 1957.

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formed by the crest of the ventricular septum and the fusion of the right and left atrioventricular valves. This defect is near the atrioventricular node and, due to injury of this node, complete heart block may follow repair. In addition, the valve deformity, which actually is part of this defect, is difficult to correct.

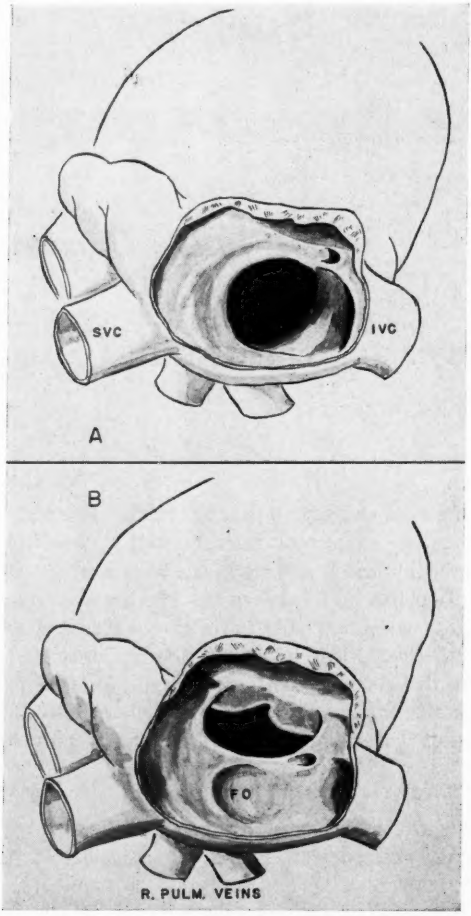


Fig. 1.—*A*, Shows a foramen ovale defect viewed from the right side, as at an operation, but with the atrial wall removed. The patient's head is to the viewer's left. The defect does not have a distinct lower margin but otherwise it offers no special problems in repair.

B, A persistent ostium primum defect with the foramen ovale normally closed. The notched aortic cusp of the mitral valve is seen through the defect. The ventricular septum is located superiorly and to the right in this picture but it is actually "lower" than or inferior to the defect when the patient is erect.

Furthermore, stitches in the ventricular septum may pull out. There are a number of difficult problems associated with attempts to repair this defect but I will not discuss them further at this time. I am concerned here with a simpler defect, the high defect which, nonetheless, offers serious hazards to repair.

High Defect.—The high defect, illustrated in Fig. 2, should be distinguished from the foramen ovale defect. It is located just inferior to the superior vena cava. The superior edge of the atrial septum in this area is a sharp crescent which forms the inferior margin of the defect but superiorly the defect has no definite margin, since there is no remnant of the septum above it. Appropriately, at least from the anatomic point of view, this defect has been called an abnormal entry of the superior vena cava into both the right and left atria.

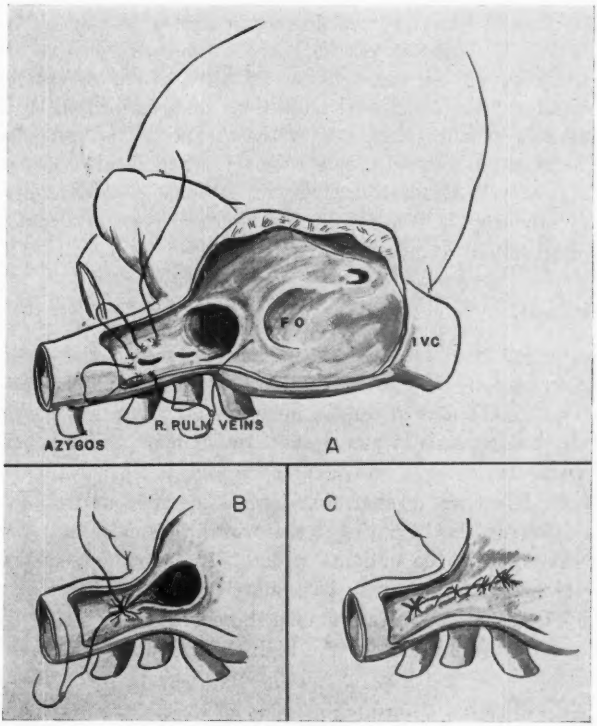


Fig. 2.—High defect of the atrial septum.

A, This shows the relationship of the defect to the septum, the fossa ovalis, the superior vena cava, and the right pulmonary veins. The orientation is from the surgeon's point of view as he stands on the right side of the operating table. The first repair stitch has been placed.

B, The first repair stitch has been tied.

C, Closure of the defect is completed and some narrowing of the vena cava is evident.

More than the vena cava and the atrial septum are involved, however, for the defect is invariably associated with a partial anomalous, pulmonary venous drainage. The veins draining the right upper and middle lobe, usually with separate openings, enter the lower part of the superior vena cava just above the defect itself. The azygos vein terminates higher than usual, entering the vena cava superiorly to these anomalous veins. This defect is superior to and separate from the site of the foramen ovale though there may be an associated defect of

the foramen ovale type, as there was in 4 of our 12 patients. To carry this association between the foramen ovale defect and the high defect farther there is, I think, a type of defect in which the band of septum between the high defect and the foramen ovale defect has absorbed resulting in a large "combined" defect. I have encountered this type only once.

The high type of atrial septal defect was described as early as 1867[18] but only occasional reports of individual cases appeared prior to our description in 1955.[9] More recently the attention of a number of surgeons has been directed to it. At the present time the pathologic anatomy of the defect is quite well understood and H. J. C. Swan of the Mayo Clinic has recently described the pathologic physiology in an excellent report on a group of carefully studied cases.[16] A search for the functional alterations he described will lead to a preoperative diagnosis in most cases. After the diagnosis has been made, surgery is indicated in most patients with this anomaly. The repair has probably been thought of as relatively straightforward with either a closed or open technique. Unfortunately, the repair is difficult and there are some special hazards to which attention has not yet been called.

CASE MATERIAL

The high defect has been the second most common atrial septal defect in our experience. Among 66 patients, all operated upon, the high defect was found 12 times. In the first 2, we did not understand the nature of the defect and only made the diagnosis in retrospect. In the third patient, however, there was a foramen ovale defect in addition to the high defect so we were forced to see that the high defect was a separate lesion. I have had no trouble recognizing it since then. Among the 12 patients, there were 7 females and 5 males, while among the remainder of the patients with atrial septal defects there were 40 females and 14 males. There may be a more nearly equal sex distribution in those with high defects than there is among those with other atrial septal defects. Age ranged from 5 to 52 years for the high defects, about the same as for the entire group.

Among the 12 patients with high defects, there were 2 operative deaths. The first death was in a 55-year-old man who died of irreversible ventricular fibrillation on the operating table—death attributable to hypothermia, the technique used for doing these open heart operations. The second death was in a 26-year-old man who died of thrombosis of the superior vena cava—a complication peculiar to the repair of this particular type of defect.

COMPLICATIONS OF REPAIR

To repair this defect we have used a row of sutures placed so that the aberrant pulmonary veins are directed into the left atrium as the defect is closed. This technique is illustrated in Fig. 2. The defect is repaired through an incision in the upper part of the atrium and lower part of the superior vena

cava. The objective of correcting both the anomalous venous connection and the septal defect is sound, of course, but the technique for obtaining this objective is not satisfactory. We have had a number of complications.

The upper half purse-string stitch may not completely close the very upper end of the repair for, in 2 cases, the catheter slipped out into the lung during postoperative catheterization as it had preoperatively. This difficulty may not be of great importance, however, for there was no evidence of a shunt in these cases postoperatively (a shunt was diagnosed when there was more than 2 volumes per cent increase in oxygen saturation between the venae cavae and the atrium). Apparently the catheter will slide out through a small crevice.

More serious have been the instances of venous thrombosis at the site of repair. Such complications have occurred in 4 of the 12 cases. All except one of these appeared as lobar pulmonary congestion in the early postoperative period indicating that the vein from the right upper lobe was occluded. Fig. 3 shows the appearance of this complication on roentgenography. In the patient illustrated, there was hemoptysis and a febrile course. Two other patients apparently were little troubled by the complication. It is not usually fatal.[5, 15] Nonetheless, when the lung recovers, the venous blood from the involved lobe must drain into the systemic venous system thus producing a left-to-right shunt; the situation that the operation was expected to correct.

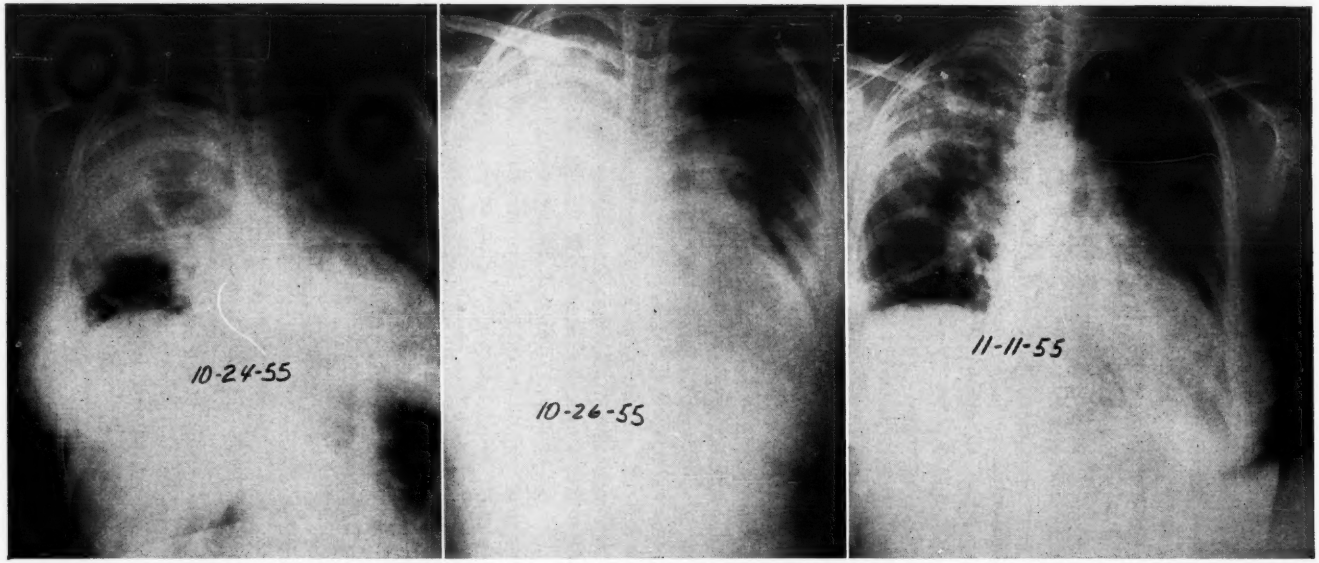
Thrombosis originating at the site of the repair may occlude the superior vena cava as well as the pulmonary veins. This happened in one of our patients and it caused his death. Apparently in trying to avoid making the channel for the pulmonary veins too narrow the vena cava was narrowed too much and it thrombosed postoperatively. Unfortunately, the azygos vein had been ligated and this closed an important potential drainage channel of the superior vena cava. This complication happened in the eleventh patient with a high defect. Probably because it had not occurred earlier, I did not consider it as a serious possibility despite the fact that narrowing of the vena cava, caused by the repair, obviously set the stage for it. When occlusion of the vena cava did occur I did not recognize it. In contrast to the chronic course usually described in the literature for patients with obstruction of the superior vena cava,[11, 12, 17] this patient passed rapidly to a fatal termination.

CASE REPORT

The patient was a 26-year-old man (Passavant Memorial Hospital No. 57-2161) known to have congenital heart disease since childhood, who underwent right heart catheterization in January, 1957, at which time a diagnosis of high atrial septal defect was made.

The defect was repaired on April 10, 1957, under direct vision with the aid of hypothermia. After completion of the repair the superior vena cava was narrow and there was cyanosis of the face while the patient was still on the operating table but blood was flowing through the vena cava.

Postoperatively, the patient did well the first day but about 36 hours after surgery he became lethargic. This lethargy gradually deepened into coma during the next 3 or 4 hours. The temperature went up to 104.6° F. and respiration failed. A tracheotomy was done and an artificial respirator was employed. The temperature was lowered with surface cooling.[10] The blood pressure fell and it was partially maintained with transfusions and



A. *B.* *C.*

Fig. 3.—Roentgenograms showing three stages in the progression and resolution of right upper lobe congestion resulting from thrombosis of the right superior pulmonary vein.

A, Beginning congestion 3 days postoperatively.

B, Massive consolidation of right upper lobe 5 days postoperatively. The patient had hemoptysis at this time.

C, Clearing of lung 3 weeks postoperatively. The patient was asymptomatic at this time.

Levophed. Congestion of the right upper lobe, indicating thrombosis of the right superior pulmonary vein, appeared on the second postoperative day (after respiratory failure and coma had developed). Spontaneous breathing did not return and he never regained consciousness. He died on the fourth postoperative day.

At autopsy examination there was a thrombosis originating at the surgical repair which obstructed the superior vena cava and the pulmonary veins from the right upper and middle lobes. There was a generalized hemorrhage into the alveolar spaces of the right upper lobe and the alveolar membranes were edematous.

The brain was soft and edematous and the subarachnoid vessels were dilated. There was a diffuse hemorrhagic necrosis of the right cerebellar lobe involving both white and gray matter. Sections through the right temporal lobe and adjacent thalamus showed necrosis of glial cells and absence of neurons.

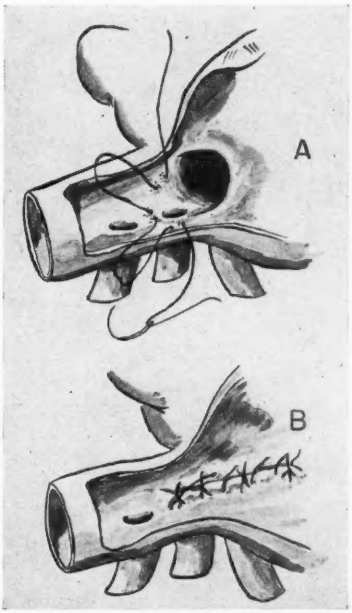


Fig. 4.—An alternative method of repairing the high atrial septal defect.
A, The first repair stitch is placed below the highest anomalous pulmonary vein.
B, Repair completed. The highest pulmonary vein is still connected anomalously to the superior vena cava but the vessels are not constricted.

IMPROVING THE TECHNIQUE OF REPAIR

Other surgeons encountering this defect have apparently used slightly different methods to repair it but it is my impression that these methods all involve narrowing of the blood channels and the introduction of foreign material in the form of sutures, plastic sponge, or both. There is probably a serious risk of thrombosis.

Perhaps the simplest way to avoid this danger and yet achieve a satisfactory repair, at the present time, is illustrated in Fig. 4. The atrial septal defect is closed but the highest abnormal vein is simply not disturbed. Our first patient

with a high defect actually had this operation because we did not know enough about the defect then to do a more complex operation. We overlooked the anomalous vein. The patient has an excellent clinical result and his heart has decreased in size. As an alternative the vein could be tied off but then, after a period of severe congestion, the venous drainage would be re-established to the vena cava anyway by way of the azygos system. The shunt through one lobe is not great and it can be safely ignored. If it seemed necessary to eliminate this shunt, the lobe could be resected.

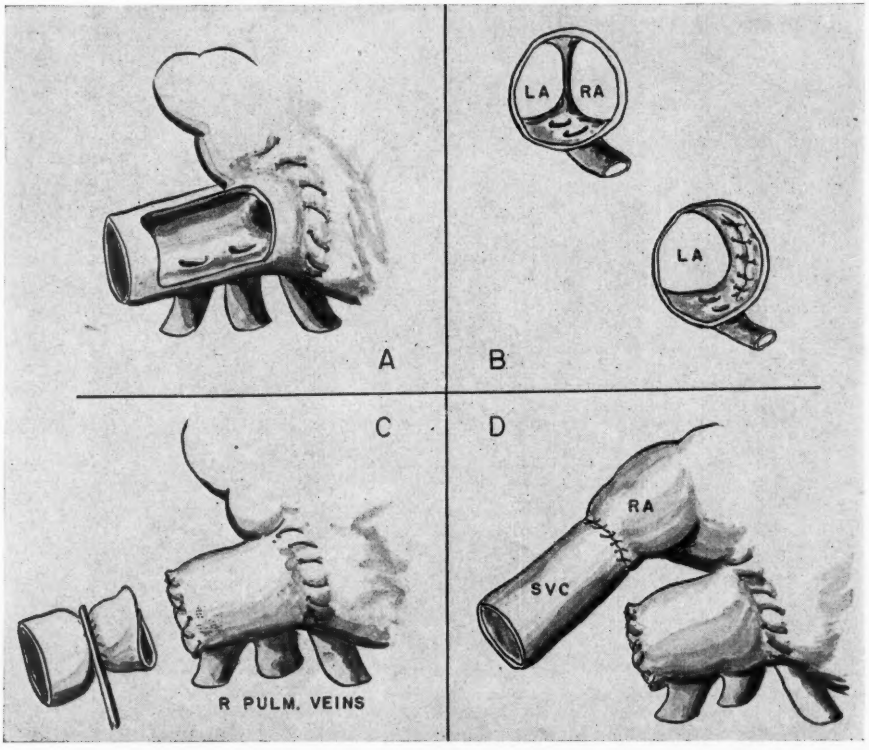


Fig. 5.—A second alternative for repairing the high defect.

A, The right atrial wall is stitched to the lower margin of the defect thus diverting the anomalous pulmonary veins and the superior vena cava into the left atrium.

B, The same step shown in *A* but viewed from above looking into the lumen of the superior vena cava.

C, The superior vena cava is divided and the distal end closed.

D, The superior vena cava anastomosed to the right atrial appendage.

Another possible way of managing the situation with achievement of complete repair is illustrated in Fig. 5. The opening of the vena cava into the auricles is revised so that the cava drains entirely into the left atrium instead of into both the right and left atria. Thus, the aberrant pulmonary veins are directed properly into the left atrium with no narrowing of their course, but the

vena cava is draining into the wrong atrium. The vena cava must then be divided above the pulmonary veins and anastomosed to the right atrium. I have not yet used this technique in patients because I considered such a venous anastomosis dangerous in itself, but we have been stimulated to study the problem of venous anastomoses in the laboratory.

ANASTOMOSIS OF THE SUPERIOR VENA CAVA TO THE RIGHT ATRIUM

Some time ago we employed Ivalon sponge blood vessels to substitute for the vena cava[1] and learned, as many others have, that grafts, or, for that matter, even anastomoses of the large veins, are very likely to become occluded. Sometimes the vessels stay open for months but the contraction of scar tissue unopposed by a significant intraluminal pressure tends to narrow the lumen and occlusion may finally occur.

The venous anastomosis which we are interested in here, between the superior vena cava and the right atrium, has been studied experimentally by Gerbode[3] who achieved good results in dogs when a careful anastomosis was made between

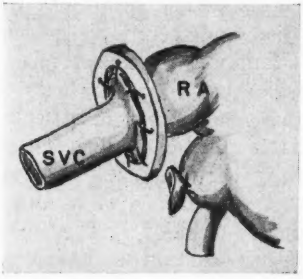


Fig. 6.—Experimental method of anastomosing the superior vena cava to the right atrial appendage. The anastomosis is attached circumferentially to a ring of Teflon.

the vena cava, just above the azygos vein, and right atrial appendage. In 7 of 10 dogs, the anastomosis remained open and some of these animals were followed for as long as 5 years.[4] In these anastomoses there was probably not much tension but as part of a treatment for the high defect tension would be unavoidable unless a graft were used. Anastomoses of various types or grafts have been employed to relieve superior vena cava obstruction in a few human cases.[6, 7, 8, 13, 14] Some of the results have been good, at least for awhile, but most of these patients were not followed long since the venous obstruction was usually due to cancer. In man, an anastomosis between the vena cava and the atrium might remain open more often than it does in dogs simply because the cava is larger but before using this anastomosis in man we hoped to learn a technique that would work regularly in dogs.

Because of our earlier experience with Ivalon grafts we have not used grafts. Instead we have concentrated on the problem of an anastomosis, done under some tension, between the vena cava, above the termination of the azygos

vein, and the right atrium.[2] In these circumstances a simple everting anastomosis failed. We then used a Teflon tube insert which was placed inside the anastomosis to hold it open. Though the actual site of anastomosis between the vena cava and the atrium could not contract down, scar tissue at either end of the tube, external to the vessel wall, narrowed the lumen and thrombosis finally occluded it. Up to the present we have had our best results with a ring of Teflon placed externally to the anastomosis which is used to hold the anastomosis open (Fig. 6). The everting anastomosis is stitched out to this ring after it has been completed. The ring is left in place so that it may hold the anastomosis open during the healing. We have used it in 12 dogs and the anastomosis has been studied with angiograms in 7. It remains open in 6 of these 7 but they have not yet been followed long enough to satisfy us.

DISCUSSION

When an anomaly, previously inoperable, becomes the proper object of surgical therapy there is a strong new stimulus to investigate all aspects of the condition. The high atrial septal defect, although a relatively uncommon disease, has become the subject of this new scrutiny. Its surgical anatomy is well understood at the present time and there is some comprehension of its embryological development. The abnormal physiology has been quite well described and, through this physiologic knowledge, new tests have been developed that permit its differential diagnosis from other atrial septal defects.

Although much of this new understanding of the defect is a result of the fact that it is now surgically correctable, the surgical repair is still not ideal. There is a propensity to the occurrence of serious complications which are clearly the result of the repair technique. Thrombosis of the anomalous pulmonary veins has been difficult to avoid, and even thrombosis of the superior vena cava may occur. The repair technique must be altered; but if it is altered in one apparently satisfactory way, it will be necessary to do a venous anastomosis (between the superior vena cava and the right atrium) which may be hazardous in itself. Perhaps the safest repair at the present time is a compromise which leaves the right superior pulmonary vein still anomalously connected to the superior vena cava.

SUMMARY

1. The high atrial septal defect can be distinguished from the foramen ovale defect and from the persistent ostium primum defect because of its unique anatomic features.

2. The complications of pulmonary venous thrombosis and thrombosis of the superior vena cava that have arisen in the repair of 12 high atrial septal defects are described.

3. In order to avoid these complications one can forego complete correction of the defect by leaving the abnormal connection of the right superior pulmonary vein to the superior vena cava. Another alternative to be explored involves anastomosis of the superior vena cava to the right atrium.

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THE ELECTROCARDIOGRAM IN OPEN HEART SURGERY

Disturbances in the Right Ventricular Conduction

Henry A. Zimmerman, M.D., Jorge Martins de Oliveira, M.D., Cid Nogueira, M.D., David Mendelsohn, M.D., and Earle B. Kay, M.D., Cleveland, Ohio

THE ADVENT of open heart surgery has raised many important questions that require elucidation. Some of these are concerned with the electrocardiographic changes during and after surgical procedures. It is the purpose of this paper to present a study of the disturbances in the right ventricular conduction which appeared after operation in patients who had undergone open heart surgery. Other disturbances in the conduction system such as auriculoventricular blocks have also been briefly considered in this work.

MATERIAL AND METHODS

Sixty-three of the first 100 patients who have been subjected to open heart surgery at St. Vincent Charity Hospital were studied by electrocardiograms taken before, during, and after the surgical procedure (Table I).

TABLE I. DISTRIBUTION OF CONGENITAL HEART DEFECTS IN 63 PATIENTS OPERATED UNDER DIRECT VISION

Interatrial septal defects	15
Pure valvular pulmonic stenosis	10
Valvular pulmonic stenosis plus interatrial septal defect	2
Valvular pulmonic stenosis plus patent ductus arteriosus	1
Interventricular septal defects	18
Pure infundibular pulmonic stenosis	2
Tetralogy of Fallot	8
Tetralogy of Fallot plus atrial septal defect	3
Infundibular pulmonic stenosis plus interventricular septal defect	4

All patients had a complete preoperative electrocardiogram (6 limb leads and precordial leads from V_1 to V_6). Recording of at least one lead during operation was undertaken in all cases. The great majority had a complete 12-lead postoperative electrocardiogram and many of them had multiple tracings in the follow-up. In a few patients who died postoperatively, it was possible to record only the limb leads due to dressings on the precordium. In order to establish a criterion for the changes indicating disturbances in the right ventricular conduction following surgery, we have divided the postoperative tracings into three groups.

From the St. Vincent Charity Hospital, Cleveland, Ohio.
Received for publication Dec. 23, 1957.

Group 1.—No changes. No alterations suggestive of disturbances in the right ventricular conduction were present.

Group 2.—First-degree changes—appearance of R′ or R′S′ in Lead V_1 but without any significant increase in the duration of the QRS complex (less than 0.015 seconds).

Group 3.—Second-degree changes—appearance of R′ or R′S′ pattern in Lead V_1, wide S waves in Lead I and wide R waves in aVR. The duration of the QRS complex is significantly increased when compared with that before surgery (equal or more than 0.02 seconds).

From now on the designations, "no changes," "first-degree changes," and "second-degree changes" will be used to indicate the alterations in the right ventricular conduction.

ANALYSIS

1. *Interatrial Septal Defects.*—All 15 patients of this group presented different types of premature beats, atrial, nodal and ventricular, during the surgical procedure, particularly during the cardiotomy. In a few cases these premature beats remained for several hours after operation, but in no case did they persist for more than 2 days (Table II).

In 14 out of the 15 cases in this series, there existed an R′ wave in Lead V_1 before the operation. This morphology is known to be a frequent finding in the electrocardiograms of patients with interatrial septal defects.[2] Following surgery in 13 of these cases, the R′ disappeared or decreased in voltage. In one case only the amplitude of this wave remained the same (Case 4). One patient in this series of interatrial septal defect had an RS pattern in the first classical precordial lead before surgery. In this case no changes were observed after the correction of the defect. Applying the criteria we have described above, we may say that of the patients with interatrial septal defects none exhibited disturbances in the right ventricular conduction after surgical correction.

2. *Valvular Pulmonic Stenosis.*—Thirteen patients presenting this malformation have undergone open heart surgery. In 10 of the cases the valvular defect was not associated with any other anomaly. The remaining 3 had other congenital defects; 2 interatrial septal defects and 1 patent ductus arteriosus. Applying our criteria we will have the following distribution for the electrocardiograms of the patients in this series. Group 1.—5 cases or 38 per cent; Group 2.—8 cases or 62 per cent; Group 3.—no cases or 0 per cent (Fig. 1).

As one can see the marked changes in the right ventricular conduction which characterize the group, 3 were absent in patients who had undergone pulmonic valvulotomy. However, in 1 case, immediately after the commissurotomy, electrocardiographic changes as described in Group 3 appeared; these were transitory and disappeared in 36 hours (Fig. 2).

In only 2 cases was the transarterial technique used; in the remaining 11 a cardiotomy through the outflow tract of the right ventricle was performed. As

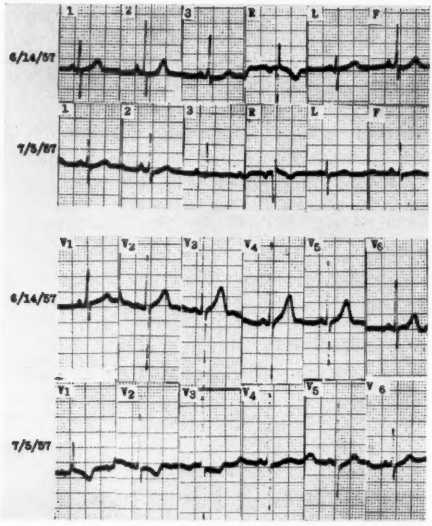

Fig. 1.—Valvular Pulmonic Stenosis. Appearance of first-degree changes after surgery.

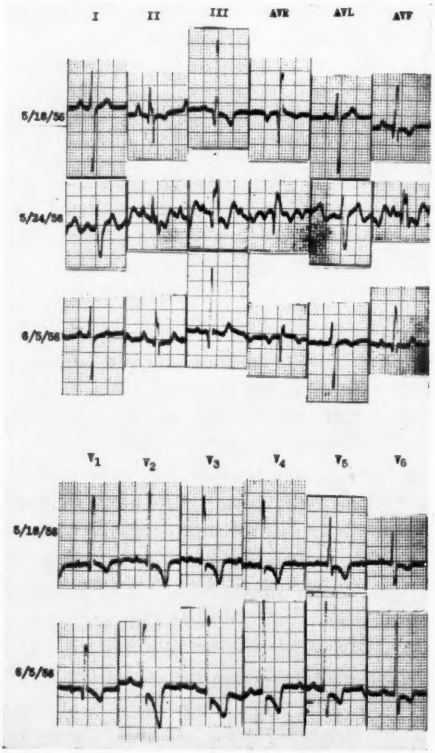

Fig. 2.—Transitory disturbances are shown in the right ventricular conduction as we can see in the record taken May 24, 1956 (a few hours after surgery). These changes disappeared and were not seen any more in the last record (June 5, 1956).

TABLE II. ECG CHANGES FOLLOWING SURGERY IN 15 PATIENTS WITH INTERATRIAL SEPTAL DEFECT

CASES	BEFORE		AFTER	
1.	R = 2.2 R′ = 8.5	S = 0.5 S′ = 2.5	R = 1.5 R′ = 8	S = 3.5 No S′
2.	R = 1.5 R′ = 5.2	S = 0.1 S = 0.5	R = 1 R′ = 3	S = 1.5 No S′
3.	R = 18	S = 4	R = 14	No S
4.	R = 2.2 R′ = 4	S = 0.1 S′ = 8.5	R = 1 R′ = 4	S = 1 S′ = 0.5
5.	R = 0.3 R′ = 10.5	S = 2.5	R = 0.1 S = 3	R′ = 8
6.	R = 0.1 R′ = 2	S = 0.2 S′ = 2	R = 0.5 No R′	S = 2 No S′
7.	R = 0.5 R′ = 5.5	S = 4	R = 0.5 R′ = 4.5	S = 3
8.	R = 1.5 R′ = 14	S = 1 S′ = 1	R = 2 R′ = 5	S = 6 No S′
9.	R = 2.5 R′ = 1	S = 1.2 S′ = 1	R = 3.3 R′ = 0.8	S = 0.3 S′ = 1
10.	R = 1 R′ = 9	S = 1.2 S′ = 5	R = 17 No R′	No S No S′
11.	R = 1 R′ = 5	S = 1.5	R = 2.2 No R′	S = 7
12.	R = 2.5 R′ = 7.5	S = 4	R = 2 R′ = 6	S = 4
13.	R = 3 R′ = 8	S = 2	R = 2 R′ = 4	S = 2
14.	R = 5 R′ = 7	S = 1.5	R = 4 R′ = 3	S = 2
15.	R = 2.5 R′ = 4.5	S = 1	R = 2 R′ = 2	S = 1.5

in the series of interatrial septal defects, our records showed several types of premature beats during operation which disappeared in the first 48 hours after surgery. In the 8 cases in which first-degree changes were present, they were seen at the moment of the valvular commissurotomy and persisted.

3. *Interventricular Septal Defects.*—In 18 patients, closure of interventricular septal defects was performed under direct vision. The comparative study of the electrocardiographic tracings showed that after surgery only 1 case could be included in Group 1, 7 patients presented first-degree changes (Group 2), and 10 exhibited alterations compatible with those described in Group 3 (Figs. 3, 4, 5).

All electrocardiographic changes appeared during the closure of the defect. Besides the already mentioned premature beats which were seen in all patients of this series of interventricular septal defects, in 2 of them, first- and second-degree A-V blocks appeared during the surgical procedure; these, however, were transitory, lasting only 2 or 3 minutes.

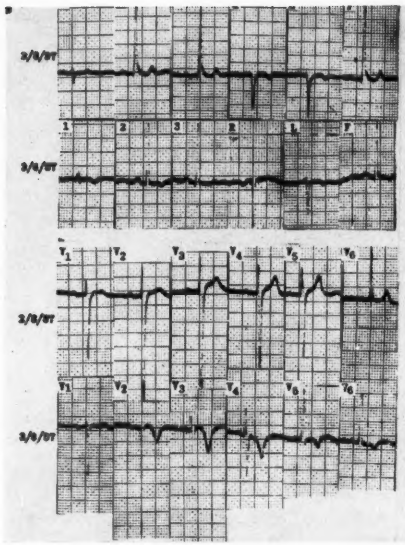

Fig. 3.

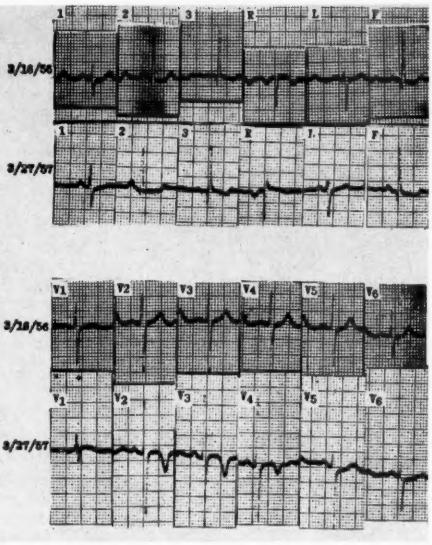

Fig. 4.

Fig. 3.—Pre- and postoperative tracings of a case of interventricular septal defect. No changes in conduction appear following the surgical procedure.

Fig. 4.—First-degree changes seen after surgery in a patient operated upon for interventricular septal defect.

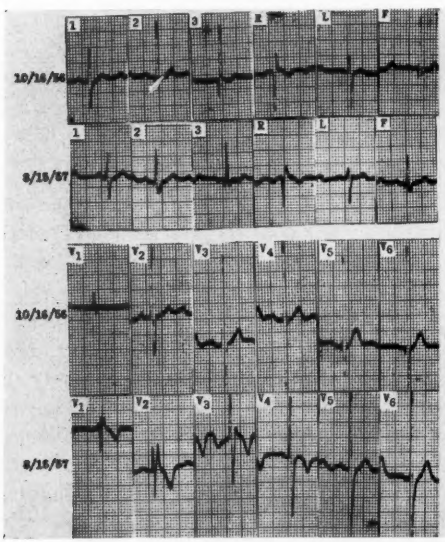

Fig. 5.

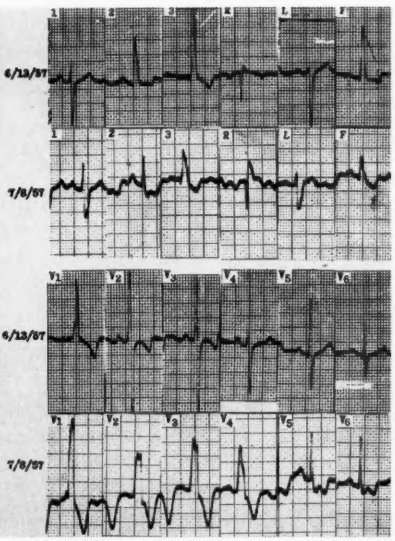

Fig. 6.

Fig. 5.—Appearance of second-degree changes following surgery in a case of interventricular septal defect.

Fig. 6.—Infundibular Pulmonic Stenosis. The postoperative tracing (July 8, 1957) shows the second-degree change pattern.

4. *Infundibular Pulmonic Stenosis.*—In the 2 cases presenting this malformation, the postoperative electrocardiograms were included in Group 3. Transient atrial and ventricular premature beats were also seen in these 2 cases during surgery (Fig. 6).

5. *Tetralogy of Fallot.*—Three out of 11 patients of this series had, also, a communication between the auricles. In 1 it was a real atrial septal defect; in the other 2 the communication was through a patent foramen ovale. One patient died immediately after the closure of the chest wall. In this case, as it is obvious, no postoperative tracing was recorded. In the remaining 10 cases (100 per cent), the electrocardiographic changes appeared either during

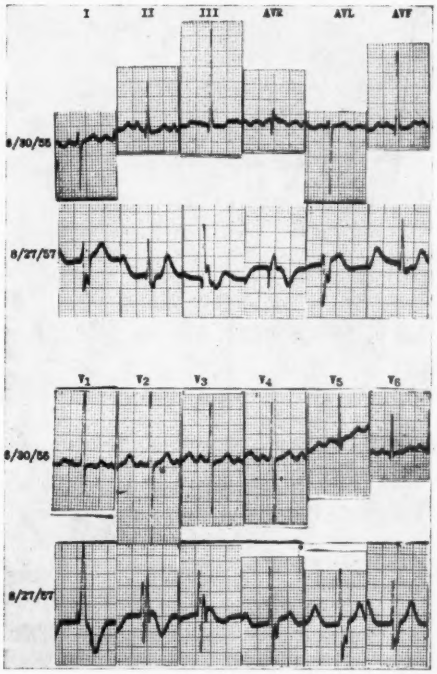

Fig. 7.—Pre- and postoperative records in a case of tetralogy of Fallot. Marked changes (second-degree) in the right ventricular conduction developed after surgery.

the resection of the pulmonary infundibulum or during the closure of the septal defect and persisted throughout the postoperative course. These changes were classified as belonging to Group 3. Premature beats were also present in all records during the surgical procedure.

In one of them a persistent complete A-V block was noticed following the closure of the interventricular septal defect. This patient, as mentioned above, died in a few minutes (Fig. 7).

6. *Infundibular Pulmonic Stenosis and Interventricular Septal Defect.*—Four patients presented infundibular pulmonic stenosis with associated interventricular septal defect, but without overriding of the aorta. In all, the

characteristic second-degree changes of Group 3 appeared either during the resection of the infundibulum or during the closure of the septal defect and persisted in the postoperative period. As in the other cases, several types of premature beats were present during surgery and persisted for some hours in the postoperative tracing (Fig. 8).

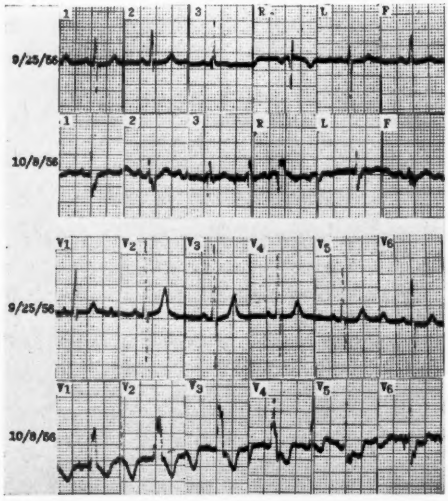

Fig. 8.—Infundibular Pulmonic Stenosis and Interventricular Septal Defect. The postoperative tracing (Oct. 10, 1956) shows the second-degree change pattern.

DISCUSSION

1. *Premature Beats.*—These changes in cardiac rhythm, in spite of its frequency, do not deserve particular consideration. In no case did they cause any disturbance to the patient; they were transitory and disappeared in a short time.

2. *A-V Blocks.*—First- and second-degree blocks were seen in a few cases, but they were transient and innocuous. They possibly occur as a consequence of edema produced by operative maneuvers close to the Aschoff-Towara node.

Complete A-V block was seen in one case of our series. We cannot say whether or not it played an important role in the mechanism of death of this patient. However, we agree with Du Shane[1] that such a disturbance in conduction when produced by mechanical injury, either of the A-V node or of the bundle of His, may cause serious effects upon the circulatory system, particularly during a procedure of such magnitude as open heart surgery.

3. *Disturbances in the Right Ventricular Conduction.*—As is shown in Table III, second-degree changes, very similar to those described in cases of right bundle branch block, appeared in all patients in which a resection of the pulmonary infundibulum was made. It was also seen in a great number of patients in whom closure of the interventricular septal defect had been performed. On the other hand, it was absent in cases of closure of interatrial septal defects or in valvular pulmonic commissurotomy (Table III).

TABLE III. ELECTROCARDIOGRAPHIC CHANGES AFTER OPEN HEART SURGERY

	NUMBER OF CASES	NO CHANGES	1ST DEGREE CHANGES	2ND DEGREE CHANGES
Atrial septal defect	15	15	0	0
Valvular pulmonic stenosis	13	5	8	0
Interventricular septal defect	18	1	7	10
Infundibular pulmonic stenosis	2	0	0	2
Tetralogy of Fallot	11	0	0	11
Infundibular pulmonic stenosis plus interventricular septal defect	4	0	0	4

These observations suggest that there is an area in the right ventricle which is always damaged during infundibular resection; it is also injured in some cases of closure of interventricular septal defects, but does not seem to be involved during valvular pulmonic commissurotomy. We reported an exception in this group. After the opening of the pulmonary valve the patient exhibited marked disturbance in conduction, but these changes disappeared within 36 hours, and probably depended upon a transitory factor involving some zone in the conduction system pathway, notably edema.

The changes in the conductive mechanism which were first seen either during the resection of the infundibulum or during the closure of the interventricular septal defect did not disappear. All patients who have been studied between 6 months and a year after the surgical procedure still had the same disturbances in the right ventricular conduction they presented during and immediately after the operation. Two regions of the conduction pathway could be responsible for the second-degree changes; the right bundle itself or some particular area located in the infundibulum. Studies performed to demonstrate the anatomic localization of the conduction system have shown that the right bundle is situated in a lower position as compared to the pulmonic infundibulum and the majority of the interventricular septal defects.[3, 4] Thus, although we admit that this right bundle branch could be affected in a few cases of infundibular resection or in closure of some defects, it would be unusual to expect its involvement in such a large number of cases. Recent studies[5, 6] indicate that some regions in the outflow tract of the right ventricle, particularly the crista supraventricularis, play an important role in the whole activation of the heart and actually represent almost all sources of the potentials responsible for the last part of the ventricular depolarization. In order to establish the localization of this "forbidden area" of the conduction system involved in our cases, a cardiotomy was performed in 5 dogs using a bypass Sigmamotor pump and a Kay-Cross oxygenator. A catheter with a special electrode was placed in the right auricle and atrial intracavitary electrocardiogram was recorded before the opening of the chest wall. During the cardiotomy, no changes in the right ventricular conduction were seen, but several types of transitory premature beats appeared. Under electrocardiographic control, stitches were placed in the higher parts of the interventricular septum and gradual resection of the infundibulum was performed. No changes in right ventricular conduction were observed until the crista supraventricularis had been cut. At this moment an electrocardiographic pattern very similar to those seen in the "so-called" right bundle branch block appeared. This was observed in 4 out of the 5 dogs

(Fig. 9). In the remaining 1, the electrocardiographic changes which developed resembled those found in Group 2 of our criteria: i.e., appearance of late R wave, but without increase in duration of the QRS complex (Fig. 10). We feel that in this 1 case the resection of the infundibular region was not complete.

One may conclude from our experiments that second-degree changes are the result of operative damage in regions involving the crista supraventricularis. This is easily accepted in cases in which the infundibulum is resected. But an explanation must be found for the changes in patients with interventricular septal defect in whom no resection is performed. The closure of high septal

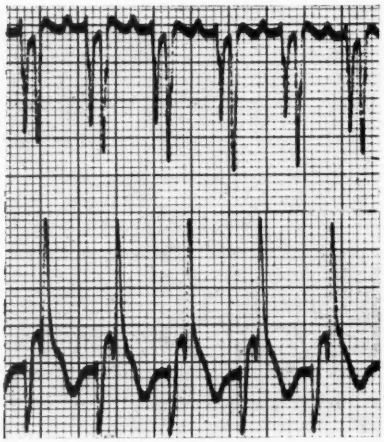

Fig. 9.

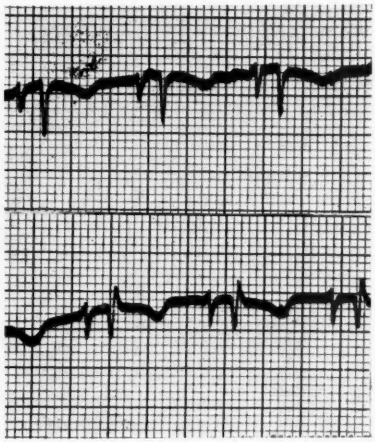

Fig. 10.

Fig. 9.—Right atrial intracavitary potential recorded in a dog before and after resection of the crista supraventricularis. Following this procedure, marked disturbances in conduction similar to those seen in cases of right bundle branch block appeared.

Fig. 10.—Right atrial intracavitary potential in a dog. After the resection of the pulmonary infundibulum, there appeared changes similar to those found in Group 2 of our criteria. In this case the infundibular resection was not complete.

defects often necessitates placing stitches in regions close to the crista supraventricularis. This may produce disturbances in the right ventricular conduction similar to those found during correction of the infundibular stenosis. On the other hand there are some cases in which the position of the defect[7] is such so as to produce damage of the right bundle branch itself when the surgical correction is performed (Fig. 11).

Thus, it seems probable that right bundle branch block pattern may result not only from involvement of the right bundle itself, but also from damage in the infundibular area, particularly if regions close to the crista supraventricularis are affected. This is not hard to accept if we consider that in many conditions in which dilation and hypertrophy of the basal portions of the right ventricle are present with consequent delay in the conduction of these areas, changes similar to those seen in Group 2 may appear.[8, 9] Thus, resection of the infundibular region or anatomic injury produced by stitches may cause delay in the conduction with development of electrocardiographic changes. We may

also add that the degree of alterations in cases of interventricular septal defects depends upon the degree of the injury produced by the operative closure of the septal hole, thus giving us first- and second-degree changes.

Another subject of importance is the effect of these disturbances in right ventricular conduction upon the patients. Although some of us believed at the beginning of this study that these changes in the conductive system could play a role in some patients in the mechanism of death, we now do not regard it as a serious possibility. For instance, in our cases of tetralogy of Fallot all 4 patients who died exhibited these conduction disturbances. However, the 7

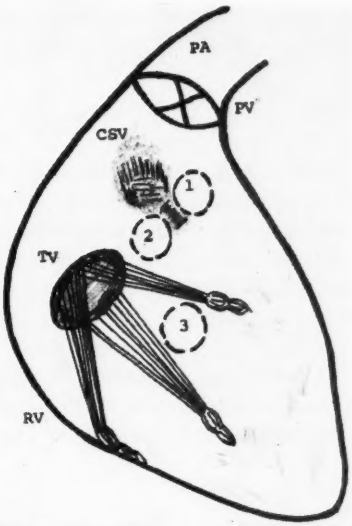

Fig. 11.—*RV*—Right ventricle. *TV*—Tricuspid valve. *CSV*—Crista supraventricularis. *PA*—Pulmonary artery. *PV*—Pulmonary valve.

Defects *1*, *2*, and *3*—Most common locations of intraventricular septal defects. As one can see, Defects No. *1* and No. *2* are situated close to the crista supraventricularis. Defect No. *3* is located near the pathway of the right branch.

who survived also had the same electrocardiographic changes. Further, patients with arteriosclerotic heart disease, Chagas disease,[10] and other cardiopathies frequently present right bundle branch block pattern without any important clinical distress. It is our opinion that the elevated mortality seen after surgery in patients operated on for pure or associated infundibular pulmonic stenosis is concerned with other factors, such as the magnitude of the operative trauma, poor hemodynamic adaptation, and electrolyte disturbance (Table IV).

SUMMARY

Disturbances in the conductive mechanism of the heart, particularly in the right ventricle, have been frequently seen in the electrocardiograms of patients operated upon for certain types of congenital cardiac malformations.

TABLE IV. CORRELATION BETWEEN MORTALITY AND ELECTROCARDIOGRAPHIC CHANGES

	NUMBER OF SURVIVALS	NO CHANGES	1ST DEGREE CHANGES	2ND DEGREE CHANGES
Atrial septal defect	14	14	0	0
Valvular pulmonic stenosis	13	5	8	0
Interventricular septal defect	12	1	4	7
Infundibular pulmonic stenosis	1	0	0	1
Tetralogy of Fallot	7	0	0	7
Infundibular pulmonic stenosis plus interventricular septal defect	2	0	0	2
	NUMBER OF MORTALITY	NO CHANGES	1ST DEGREE CHANGES	2ND DEGREE CHANGES
Atrial septal defect	1	1	0	0
Valvular pulmonic stenosis	0	–	–	–
Interventricular septal defect	6	0	3	3
Infundibular pulmonic stenosis	1	0	0	1
Tetralogy of Fallot	4	0	0	4
Infundibular pulmonic stenosis plus interventricular septal defect	2	0	0	2

A study of 63 patients who had open heart surgery was undertaken in order to evaluate the incidence and meaning of these disturbances.

A correlation carried out with findings obtained from experimental studies in dogs demonstrated that the changes in the conductive system are related either to the right branch bundle itself, or to the crista supraventricularis and surrounding areas.

It was concluded that these disturbances in conduction were not responsible for the elevated mortality in the group of pure and associated infundibular pulmonic stenosis, but that the deaths in this group depended upon other factors such as operative trauma, hemodynamic inadaptation, and electrolyte disturbances.

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STUDIES OF HEMOLYSIS WITH A PLASTIC-SHEET BUBBLE OXYGENATOR

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DELETERIOUS effects on blood in a pump-oxygenator result from a variety of physical actions on the blood. The measurement of hemolysis, expressed by the increase in the value for hemoglobin in the plasma, is used as an index of the degree of undesirable effects exerted on the blood. For the index to reflect properly the deleterious characteristics of a pump-oxygenator, determinations of hemoglobin in the plasma probably should be done with the blood circulating in the apparatus only, while the arterial line is connected to the venous line directly. A stated quantity of blood should be circulated at a stated rate of flow.

The plastic-sheet bubble oxygenator, described by Gott and co-workers,[1] was chosen for this study and comparative determinations of the hemoglobin content of the plasma were carried out with and without an animal in perfusion.

METHODS

For each of the experiments 2,000 c.c. of recently drawn, heparinized blood was obtained from 1 or 2 healthy mongrel dogs of 16 to 30 Kg. in weight by cannulation of the left common carotid artery. For the cannulation, the dogs were anesthetized with 8 to 15 c.c. of a 2.5 per cent solution of thiopental sodium. Five minutes before phlebotomy, heparin was injected intravenously in amounts of 3 mg. per kilogram of body weight.

Dogs which underwent perfusion were healthy mongrels of 8 to 12 Kg. The method for whole-body perfusion was identical to that described by Penido and co-workers.[2]

Plastic-sheet bubble oxygenators* were used in all experiments. One pump of a roller occlusive type† was used as an artery pump, providing a flow of blood of 550 c.c. per minute. The venous return into a venous reservoir was obtained by gravity. Oxygen flow rates of 2,000 c.c. per minute were used in all experiments, except in one, in which compressed room air was used at the same rate of flow. The connections were made by plastic Tygon tubing with an internal diameter of ½ and 5/16 inch and stainless steel connectors. The venous reservoir was made of Lucite.

From The Mayo Clinic and Mayo Foundation, Rochester, Minn.
The Mayo Foundation, Rochester, Minnesota, is a part of the Graduate School of the University of Minnesota.
Received for publication Jan. 16, 1958.
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†Rotaflex, Cole Parmer Co., Chicago, Ill.

In the first group of experiments, 2,000 c.c. of recently drawn, heparinized donor blood was circulated in the pump-oxygenator system for 1 hour or 2 hours. In one of these experiments compressed room air was used instead of oxygen. Samples of blood (10 c.c.) for the determination of the hemoglobin content of the plasma were taken before the blood was introduced into the oxygenator, and then from the venous line every 15 minutes during the run.

In the second group of experiments, a dog underwent whole-body perfusion for 1 hour with the same rates of flow for blood and oxygen as before. Samples for the determination of hemoglobin in the plasma were taken before and every 10 minutes during the perfusion. In addition to this procedure, 2,000 c.c. of the blood was circulated in the pump-oxygenator system for periods of a ½ to 1 hour before or after the blood was used for whole-body perfusion.

The samples of blood were carefully collected in test tubes containing 2 drops of heparin, and were centrifuged for 20 minutes at a speed of 1,800 r.p.m. The supernatant plasma was pipetted off. Determinations of plasma hemoglobin were carried out according to the method of Shinowara[3] in the Beckman spectrophotometer. This method was found to be advantageous over other methods because it allows accurate determinations of the content of hemoglobin in the plasma to ranges as low as 5 to 10 mg. per 100 c.c.*

RESULTS

Fig. 1 gives the mean values for plasma hemoglobin as found in 8 experiments, when 2,000 c.c. of blood was circulated in the closed pump-oxygenator circuit for 1 hour or 2 hours. The resulting curve shows a constant rise, reaching an average of 100 mg. of hemoglobin per 100 c.c. of plasma within 30 minutes, of 350 mg. within 60 minutes, and then leveling off gradually toward the end of 120 minutes, reaching a value of 500 mg. of hemoglobin per 100 c.c. of plasma.

An essentially similar curve was obtained when compressed room air was used in the oxygenator instead of oxygen at the same rate of flow (Fig. 2).

To define the role of the pump and the oxygenator independently in the production of hemolysis, the oxygenator was removed from the circuit during a portion of the experiments and was replaced by a glass container. Regardless of whether this was done in the first or second half of the experiment, it could be seen that the pump and tubing accounted for an increase of hemoglobin in the plasma of only about 25 mg. per 100 c.c. within 60 minutes, whereas a typical increase up to 275 mg. per 100 c.c. was produced within 60 minutes by the oxygenator (Fig. 3).

When this pump-oxygenator system was used under the same conditions to perfuse a dog for 1 hour, changes in values for plasma hemoglobin proceeded in an entirely different manner. If marked hemolysis was not present before perfusion was begun, the value for hemoglobin in the plasma increased gradually to 20 to 30 mg. per 100 c.c. within 1 hour (Fig. 4).

When perfusion of an animal was begun after a preliminary run "in vitro" had produced a value of 125 mg. of hemoglobin per 100 c.c. of plasma, a rapid decrease to 12.5 mg. of hemoglobin per 100 c.c. was observed in the

*We are indebted to Dr. E. J. Baldes of the Mayo Foundation and to Dr. T. T. Myre of Memphis, Tenn., for establishing this method for the determination of plasma hemoglobin in this laboratory.

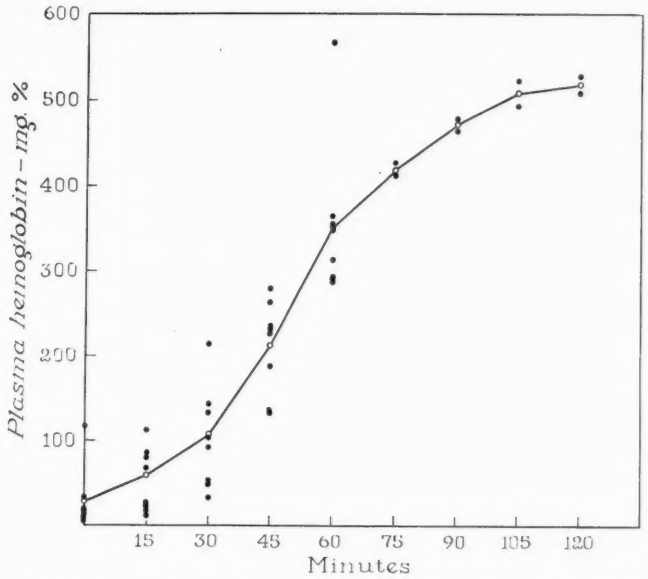

Fig. 1.—Mean values for hemoglobin in the plasma when 2,000 c.c. of blood was circulated in the pump-bubble oxygenator system for 2 hours. Pump flow: 550 c.c. per minute; oxygen flow: 2 L. per minute.

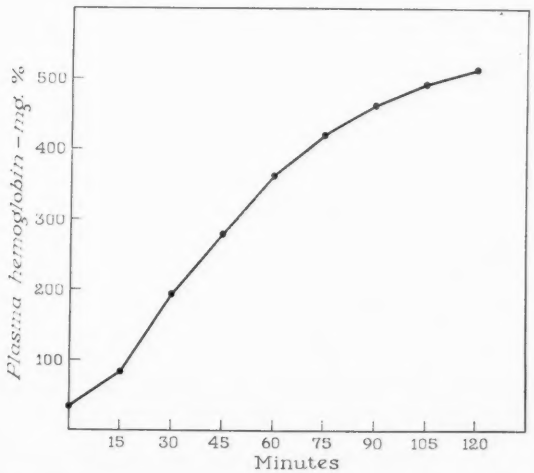

Fig. 2.—Concentrations of hemoglobin in the plasma when 2,000 c.c. of blood was circulated in the pump-bubble oxygenator system for 2 hours. Pump flow: 550 c.c. per minute; compressed room air flow: 2 L. per minute.

next few minutes, and during the actual perfusion the plasma hemoglobin increased only gradually to 40 mg. per 100 c.c. in 1 hour. As 2,000 c.c. of the blood was circulated in the closed pump-oxygenator system after perfusion was terminated, a prompt increase in hemolysis occurred (Fig. 5).

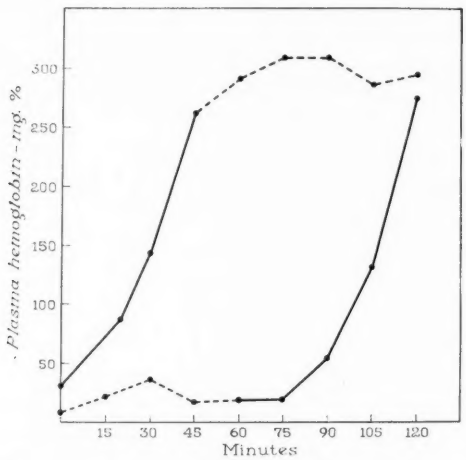

Fig. 3.—Concentrations of hemoglobin in the plasma when 2,000 c.c. of blood was circulated in the pump-bubble oxygenator system (solid lines) or pump and tubing system alone (broken lines), the oxygenator being replaced by a glass container. Pump flow: 550 c.c. per minute; oxygen flow: 2 L. per minute.

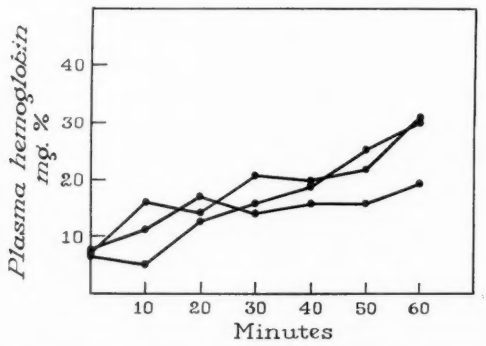

Fig. 4.—Concentrations of hemoglobin in the plasma during whole-body perfusion of dogs (9, 10.7 and 12 Kg.) with 2,000 c.c. of blood from the pump-bubble oxygenator for 1 hour. Pump flow: 550 c.c. per minute; oxygen flow: 2 L. per minute.

To investigate the part played by a living organism in this phenomenon, a comparative experiment "in vitro" was done: a glass container with 800 c.c. of recently drawn, heparinized and unhemolyzed blood was added to the pump-oxygenator circuit after some degree of hemolysis had taken place in 2,000 c.c. of blood during a run of 30 minutes under the usual conditions (Fig. 6).

When fresh blood entered into the circuit a sharp decrease in the concentration of hemoglobin in the plasma was seen, similar to that depicted in Fig. 5. However, in its further course this curve was significantly different.

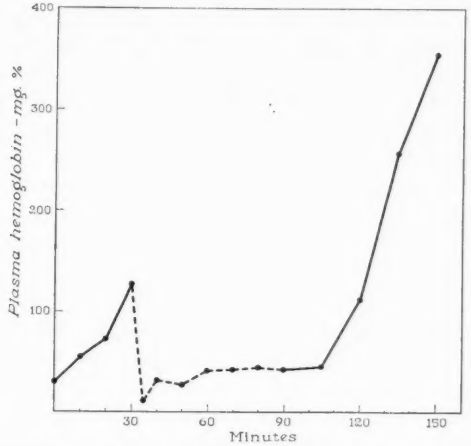

Fig. 5.—Concentrations of hemoglobin in the plasma when a dog (9 Kg.) was perfused for 1 hour (broken line); 2,000 c.c. of blood was circulated in the pump-bubble oxygenator 30 minutes prior and 60 minutes after perfusion of the dog (solid line). Pump flow: 550 c.c. per minute; oxygen flow: 2 L. per minute.

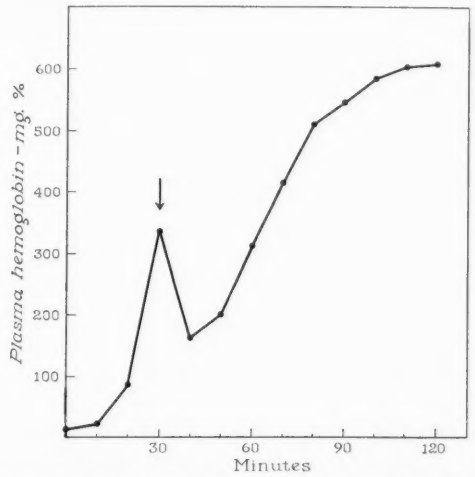

Fig. 6.—Concentrations of hemoglobin in the plasma when 2,000 c.c. of blood was circulated in the pump-bubble oxygenator for 2 hours. After 30 minutes a beaker containing 800 c.c. of fresh, unhemolyzed blood was introduced into the circuit (arrow). Pump flow: 550 c.c. per minute; oxygen flow: 2 L. per minute.

In the experiment "in vitro" a steady and rapid increase in hemolysis took place, as would be expected in the use of this pump-oxygenator, in contrast to the low concentrations of hemoglobin in the plasma maintained for 1 hour when a living organism is in the circuit.

COMMENT

In all experiments in which blood was circulated in the pump-oxygenator only, a steady and rapid increase in the concentrations of hemoglobin in the plasma up to 350 mg. per 100 c.c. within 1 hour and 500 mg. per 100 c.c. within 2 hours was observed, whereas during the perfusion of a dog the concentrations of hemoglobin in the plasma did not increase to more than 30 mg. per 100 c.c. In the interpretation of the experiments depicted in Figs. 5 and 6, we are aware of the fact that no quantitative estimations can be made. The initial decrease in both curves is probably due in part to a dilution effect and in part to inadequate mixing of the fresh blood with the hemolyzed blood. The different behavior in the further course of these two curves, however, suggests that the living organism actively removes plasma hemoglobin from the system, or modifies its rate of production in the apparatus. The former is the more probable.

Gott and co-workers[1] found that this oxygenator produces an average increase of 77 mg. of hemoglobin per 100 c.c. of plasma (range from 8 to 185 mg. per 100 c.c.) when dogs are perfused for 1 hour. This is supported by our data. DeWall and co-workers[4] reported values for hemolysis of 33 mg. of hemoglobin per 100 c.c. of plasma (range from 6 to 80 mg. per 100 c.c.) in the clinical use of their oxygenator for the perfusion of man. Recently, Bücherl[5] published results of experiments "in vitro" in which he passed oxygen through human donor blood according to the bubble-oxygenator principle. He noted values for hemoglobin in the range of 300 mg. per 100 c.c. of plasma after 1 hour, increasing to the range of 500 mg. per 100 c.c. in the second hour, which is in close agreement with our findings.

It seems that the jets of oxygen entering the blood at the bottom of the oxygenator chamber are the major factors in the production of such intensive hemolysis. In evaluating different arrangements of the oxygen disperser, Gott and co-workers[1] noted extremely high hemolysis (an increase of 971 mg. of hemoglobin per 100 c.c. of plasma) and a 100 per cent mortality rate among 5 dogs when the venous inflow was below the level of the oxygen disperser and oxygen emitted under high pressure through only a few fine holes. Bücherl[5] observed an increase in hemolysis when oxygen flows were higher and, also, when an antifoam substance was added to the blood. This latter fact suggests that the passage of gas through a massive column of blood with the resulting turbulences is the mechanism which causes high hemolysis, and that the foaming does not do so.

It is unlikely that oxygen in itself causes hemolysis, since we found essentially the same values when either pure oxygen or room air was used.

Several factors exert an influence in the proportion of hemoglobin liberated from the plasma by trauma. The percentage volume of red blood cells was found to have a decided influence. The condition of the donor blood, especially if it was drawn from more than one donor, or was stored, certainly may make the blood more or less susceptible to hemolysis. This is obvious not only in increased concentrations of hemoglobin in the plasma of the control sample; there is also the possibility that these erythrocytes are more likely to

be affected by trauma. It is known that cohesion of erythrocytes, spheroidicity, changes in the strength of the membrane and heating to 52° to 58° C. definitely increase the mechanical fragility of erythrocytes.[6]

Consideration should be given to the possibility that large amounts of plasma hemoglobin are removed from the blood by the subject during whole-body perfusion if the pump-oxygenator produces high hemolysis. The mechanism of elimination of hemoglobin from the plasma in the blood stream has been studied by many investigators.[7-13] Three possibilities are recognized. After injection of solutions of hemoglobin into the blood stream, the plasma hemoglobin is quickly picked up by the cells of the reticulo-endothelial system, where it is broken down and transformed to bile pigments. This is the only mechanism involved if the concentration of hemoglobin in the plasma stays below a certain level. Above this level, hemoglobin is also excreted through the kidneys. A third way for the elimination of intravascular free hemoglobin was suggested by Fairley.[8] He showed that at higher levels the hemoglobin could be catabolized directly in the blood stream with the formation of methemalbumin.

The renal threshold for hemoglobin in human beings is in the neighborhood of 135 to 250 mg. per 100 c.c. of plasma.[9, 14, 15] For dogs it has been found in the range of 100 to 210 mg. per 100 c.c.[16-20] Once hemoglobinuria has started, it may continue until the concentration has decreased to 30 to 50 mg. per 100 c.c. of plasma.[16] The renal clearance of hemoglobin has been found to lie between 3 and 6 mg. per 100 c.c. of plasma per minute, if the concentration of hemoglobin is 250 mg. per 100 c.c. of plasma.[21] During whole-body perfusion these relationships may be altered, for renal blood flow and renal function are closely related to the rate of perfusion and aortic mean pressure.[22]

Bethard and Plzak[23] injected radioactive labeled solutions of hemoglobin into nephrectomized rats and found that the rates for clearance of the injected material from the plasma in the nephrectomized animals were 30 to 40 per cent of those in normal controls. Flink[10] estimated that after the injection of 1 Gm. of hemoglobin per kilogram of body weight in dogs, 60 to 75 per cent was destroyed by the reticulo-endothelial system.

The reticulo-endothelial system certainly is capable of clearing large amounts of hemoglobin from the plasma, as can be seen in patients who have "acholuric" jaundice, in which the total hemoglobin of the body is disposed of within a few days and up to 10 Gm. of hemoglobin is destroyed in 1 day without the appearance of hemoglobin in the urine. The reticulo-endothelial cells of the spleen, liver, and bone marrow probably are the main foci for the destruction of hemoglobin, but the reticulo-endothelial system of the whole body takes part in the process, so that large amounts of free hemoglobin are cleared from the blood stream within seconds or minutes.

An increase in bilirubin in the plasma should be expected and has been proved to occur within 30 minutes after the injection of solutions of hemoglobin into human beings.[7, 9] The life span of bilirubin molecules in the plasma is thought to be approximately 90 minutes.[11]

The above-mentioned data allow the following interpretation of the fate of plasma hemoglobin in our experiments.

Quantities of up to 350 mg. of free hemoglobin per 100 c.c. of plasma are produced in 1 hour by the pump-oxygenator studied. If there is a subject in perfusion during this time, the failure of free hemoglobin levels in the plasma to rise is most likely caused by the action of the reticulo-endothelial system. Since concentrations of hemoglobin remained below 50 mg. per 100 c.c. of plasma, excretion through the kidneys is unlikely, especially if a reduction in renal blood flow occurs during perfusion.

If it is assumed that the animal removes hemoglobin from the blood at a rate of 300 mg. per 100 c.c. of plasma per hour, the clearance of hemoglobin would be 5.0 mg. of hemoglobin per 100 c.c. of plasma per minute.

The clinical sequelae of hemoglobinemia of moderate degree have been described as consisting of chills, fever, abdominal cramps, and vomiting.[9] Only at high levels would hemoglobinuria result and enhance the dangers of anuria, either by obstruction of the renal tubules by casts and debris of hemoglobin or its derivatives,[24] or on the basis of renal vasoconstriction induced by the presence of extracorpuscular hemoglobin.[25] Hyperbilirubinemia may lead to jaundice if 300 to 500 c.c. of blood is completely hemolyzed within a few hours.[13]

It seems that hemoglobinemia and an increased uptake of hemoglobin as encountered in our experiments would cause little damage to the subject, except perhaps for a certain degree of fever. However, the increase in hemoglobin in the plasma is an index of deleterious action on the blood, and other factors may be of greater clinical importance. It can be expected that, with the liberation of hemoglobin from red blood cells, a corresponding amount of red-cell stromata is released. This poorly defined substance has been said to be responsible for more serious sequelae of hemolysis. Barratt and Yorke[23] described the lethal character of red-cell stromata after these workers injected relatively small amounts of the material into rabbits, whereas injection of purified solutions of hemoglobin, even in large amounts, produced no or only slight effects. Peripheral vascular dilatation resulting in hypotension, bradycardia, and impaired cardiac conduction with heart block have been attributed to adenosine triphosphate and related compounds set free within 15 to 30 seconds when red blood cells are destroyed. Deyrup[27] showed that quantities of adenosine derivatives from about 3 to 18 per cent of the erythrocytes hemolyzed in a given sample of blood produce significant hypotensive states, and that postoperative shock has been recognized as a possible complication after hemolysis.[28-32]

Disturbances in the blood-clotting mechanism also have been attributed to hemolysis. Barratt and Yorke[26] thought these disturbances to be due to intravascular separation of fibrin in the blood vessels. Fogelman and co-workers[33] reported marked prolongation of the coagulation time in 5 of 9 animals when high degrees of hemolysis were produced.

SUMMARY

It is suggested that the hemolysis which occurs in the application of a pump-oxygenator system should be tested for the pump-oxygenator circuit proper without a subject in perfusion.

Comparative experiments with a plastic-sheet bubble oxygenator have been carried out. The pump-oxygenator system within 1 hour produced hemolysis resulting in a concentration of 350 mg. of hemoglobin per 100 c.c. of plasma and 500 mg. per 100 c.c. within 2 hours, when 2,000 c.c. of recently drawn, heparinized canine blood was circulated at a pump rate of flow of 550 c.c. per minute and a flow of oxygen of 2 L. per minute. With an animal in the circuit, the concentration of hemoglobin did not increase more than 30 mg. per 100 c.c. of plasma in 1 hour.

The main factor in the production of hemolysis in this apparatus may be the oxygen jets which cause turbulent flows in the bubble chamber.

The resulting physiopathologic consequences are discussed. It is assumed that during whole-body perfusion the reticulo-endothelial system continuously removes hemoglobin in the plasma from the blood stream at approximately the same rate as the hemoglobin is produced. The rate of clearance was established to be approximately 5 mg. per 100 c.c. of plasma per minute.

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EXPERIMENTAL CORONARY ARTERIOGRAPHY

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IN THE TREATMENT of coronary disease, improvement of flow through occluded vessels might in certain circumstances be possible by surgical restoration of patency. This approach is being investigated in many laboratories and has had limited clinical trial. Basic to any promise of success is a method for the accurate delineation of occluded and open vessels. Several methods for experimental[1-10] and clinical[4, 11, 18] coronary arteriography have been designed. These, in the main, utilize radiopaque material introduced into the first part of the aorta by direct puncture[11, 13, 15, 16] or via a tube in a peripheral artery. Temporary occlusion of the aorta by an inflated balloon increases the penetration of the contrast material into the coronary system.[15]

The technique to be described below was not designed primarily for clinical use, but rather to attain the best possible delineation of fine coronary branches for studies of the evolution of collateral circulation. Uniform distribution of contrast medium in high concentration within the coronary arteries has been obtained by a method of retrograde injection, developed in earlier work on other vascular beds,[19] which provides nearly complete displacement of blood by contrast medium during the instant of taking the radiogram. The need to expose the heart for the injection of contrast material is not considered a disadvantage since the method is intended for use in connection with surgical procedures upon the coronary vessels.

MATERIAL AND METHODS

Animals.—The experiments were performed upon 31 adult male and nonpregnant female mongrel dogs, weighing between 18 and 31 Kg.

Technique.—The animals were anesthetized with Nembutal given intravenously. Lung inflation was maintained by intratracheal tube. The left chest was opened through the fifth interspace and the heart was exposed through a longitudinal incision in the pericardium, anterior to the phrenic nerve.

A small primary or secondary branch of the anterior descending coronary artery leading over the surface of the left ventricle, not far below the atrioventricular sulcus, was isolated and incised for cannulation (Fig. 1). Distal

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This investigation was supported by grants from the Life Insurance Medical Research Fund, and the National Institutes of Health, Public Health Service (H-1984).

Received for publication Jan. 27, 1958.

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ligation was required because of free arterial back-bleeding. Note was taken of the development of any myocardial cyanosis. The open end of a heparin-filled polyethylene tube was introduced and passed proximally to the origin of the cannulated branch and secured with a fine silk tie and one or two fine stitches. The tubes used for cannulation ranged in size from PE 60 to PE 190 (Intramedic scale—outer diameters 1.22 to 1.70 mm.).

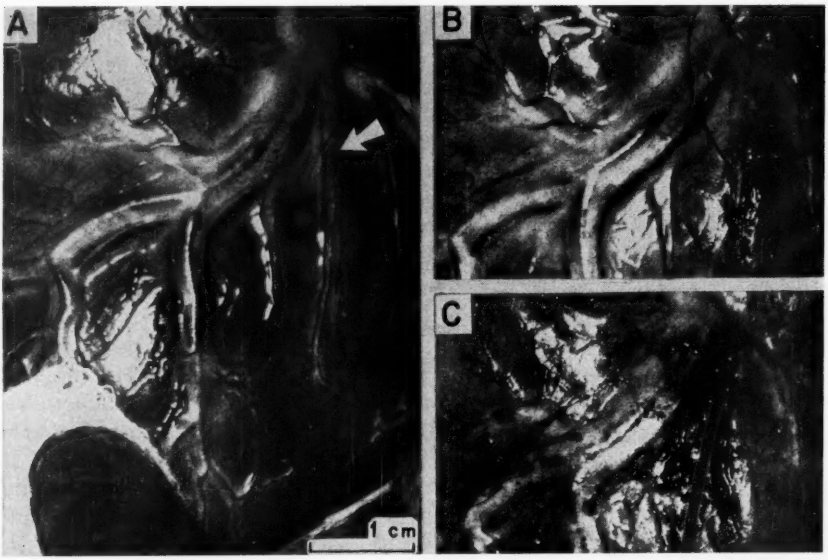

Fig. 1.—Photographs of stages in the cannulation of a coronary branch for arteriography in a dog.

A, Left surface of the heart showing upper portion of the anterior descending coronary artery, its branches, and accompanying veins close to the atrioventricular sulcus. The left auricular appendage is shown in the right upper corner of the photograph. At the left lower corner a gauze-covered finger makes downward pressure to expose the branch to be cannulated (*arrow*). *B* shows the coronary branch during its isolation and *C* the cannula tied into the vessel.

Tolerance to the injection of undiluted contrast material into the left coronary system was tested in three preliminary experiments by repeated injection of 10 c.c. volumes of Hypaque* while the exposed heart was kept under observation.

Angiograms were done in 18 dogs. In these experiments the polyethylene tube was led out through the pericardium and the sixth interspace anteriorly or posteriorly and the chest was closed for transfer of the animal to the X-ray Department.† The left chest wall was placed directly upon the x-ray cassette. Standard radiological equipment and x-ray film‡ were used. Ten cubic centimeters of contrast medium was injected rapidly by hand (3 to 5 seconds) using a 10 c.c. syringe and a one-meter length of wide tubing connected to the

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†We are grateful to Dr. Felix Fleischner, Chief of the Department of Radiology, for his advice and cooperation, and to Mr. Louis Baizman for technical assistance.

‡Eastman Kodak Blue Brand Film.

fine polyethylene tube. Just before completion of the injection the radiogram was taken at 46-60 kV., 400 mA., and $\frac{1}{60}$ second. The focus to film distance was 90 cm. An intensifying screen was used, but no grid. One to 5 arteriograms were taken in each dog at intervals of 5 to 15 minutes, each exposure with a separate injection of contrast material.

For stereoscopic views, additional arteriograms were taken with the tube moved 3 to 5 cm. horizontally.

A few serial angiograms* were taken with a rapid cassette changer and an impulse timer, using 90 kV., 700 mA., and 1.5 milliseconds. Up to 20 exposures were obtained at a rate of 3 per second, before, during, and just after the injection of contrast material.

The standard technique for a single angiogram prior to a procedure upon a coronary artery is completed by removal of the catheter and ligation of the incised branch. Most of the present experiments were terminated at this point. A number of the dogs were sacrificed at once and the hearts excised for detailed stereoradiographic study of the coronary pattern. The coronary arteries were injected with Micropaque (40 per cent suspension in 2.5 per cent solution of gelatin in water) introduced into the left coronary system through the cannulated branch after suture closure of the left coronary ostium, and into the right coronary system through a cannula in the right coronary ostium.

In a few experiments the tube was left in place, its outer end tied and covered with a dressing. The lumen was cleared with a small quantity of heparin at intervals ranging from 1 day to 1 week, and arteriograms were taken weekly. Aseptic technique was used whenever the experiments were to be carried beyond the day of cannulation.

Electrocardiographic recording was carried out during the cannulation and injection of contrast material in 7 dogs, and the myocardium was subsequently studied histologically for evidence of injury due to ischemia or chemical trauma. These tests were carried out with tube sizes PE 90 to 100.

RESULTS

The injection of three 10 c.c. doses of Hypaque in rapid succession produced no change in heart action in any of the 3 dogs tested except for a transitory slowing in the rate. A fourth injection given immediately thereafter led to ventricular fibrillation in 2 of the dogs.

The procedure was tolerated well in the angiographic experiments. Intervals of 5 minutes or longer were maintained between injections of contrast material and 17 of 18 dogs received 42 injections without evident ill effect. One dog died a minute or two after completion of an initial injection, probably of ventricular fibrillation. Anesthesia in this case had been maintained for something over 3 hours and there was evidence that the dog was anoxic at the time of injection.

Electrocardiographic observations demonstrated T-wave depression beginning 2 seconds after the start of the injection. By the end of the injection

*We are grateful to Dr. Edward B. D. Neuhauser of the Children's Medical Center, Boston, for permission to use the equipment of his department, and to Mr. Eric Hammond for his technical assistance.

(3 to 5 seconds) the T waves were strongly negative. At this time the heart rate slowed somewhat. Return toward normal began within 1 second after termination of the injection and was complete within 30 to 60 seconds. Similar changes were seen when physiologic saline solution was injected instead of Hypaque. Ventricular fibrillation terminated a series of injections in 2 of these electrocardiographic control experiments. In both cases, this occurred during a period of induced general anoxia, produced by reduction in pulmonary ventilation until electrocardiographic evidence of myocardial anoxia appeared. In one, the injection period was also prolonged. Defibrillation was readily accomplished by electric shock.

Hand injection of contrast medium through tubes smaller than PE 90 proceeded too slowly to provide the desired concentration within the coronary arteries. Although the medium was distributed well through the branches of the left coronary artery the photographic contrast was poor. Injection through tubes PE 90 to 100 was rapid enough to permit quite complete displacement of blood by contrast medium and no advantage was gained by using larger tubes. The sacrifice of a coronary branch just able to accommodate a PE 90 to 100 tube produced no color change on the surface of the heart in most experiments. In 2 dogs, the cannulated branch was unusually distant from neighboring branches and a small area of cyanosis appeared. The cyanosis had largely cleared by the time of closure of the pericardium. Cannulation produced no change in cardiac rhythm and only an occasional slight and transitory downward deflection in T wave. However, a small intramural infarct was usually found upon histologic examination of the tissue supplied by the sacrificed branch. The zone of myocardial injury did not in any case exceed 5 to 10 mm. in length and 3 to 5 mm. in greatest transverse diameter. Histologic examination of coronary vessels and myocardium away from the site of ligation showed no evidence of injury.

The post-mortem angiograms showed that the in vivo delineation of the left coronary system was complete within the size range of the branches demonstrated by the method (Fig. 3) and confirmed the localization and identification of the branches given by the in vivo stereo-angiograms. The smallest vessels clearly visualized in vivo measured 0.2 mm. in diameter in the post-mortem specimen.

The standard single-exposure arteriograms regularly showed extensive contrast filling of the left coronary system; the distribution of the circumflex and septal arteries being shown as well as that of the anterior descending coronary artery. Definition was good. Sometimes the vessel outlines, either at the apex or at the base of the heart, were not entirely sharp, but quite often the entire distribution of the left coronary artery was sharply visualized so that branches of the third order could readily be made out (Fig. 4). The pattern of the left coronary system was reproduced well in repeated angiograms, with some variation only in the extent of outlining of branches of the third order. The cannulated branch beyond the point of ligation was not visualized in the initial angiograms.

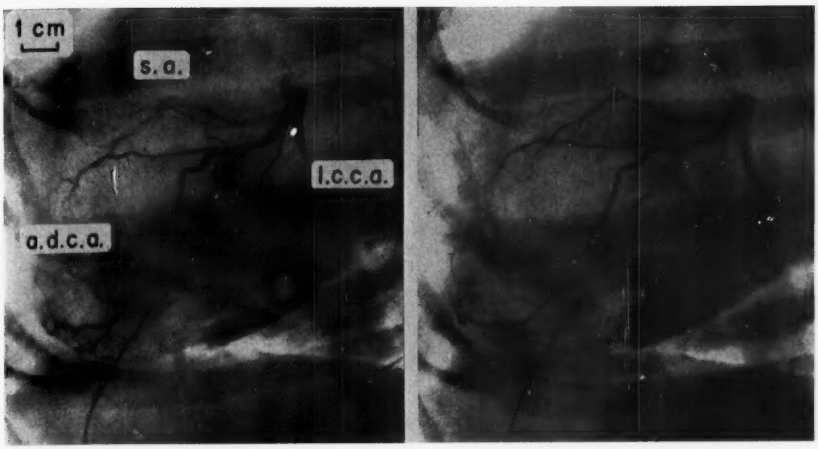

Fig. 2.—Stereoscopic coronary arteriograms reduced in size. The second exposure is obtained with a second injection of contrast medium.

The polyethylene cannula enters a small coronary branch; a slight blur of extravasated contrast medium in the region of dissection indicates the place of cannulation. The polyethylene tube runs in a loop in the left chest; it has moved slightly with the respiration of the animal between exposures and therefore gives a false impression of its depth location.

The left circumflex (*l.c.c.a.*), the anterior descending (*a.d.c.a.*), and the septal artery (*s.a.*) are indicated in the illustration. No contrast material is seen in the aortic bulb in either exposure and the right coronary artery is not visible.

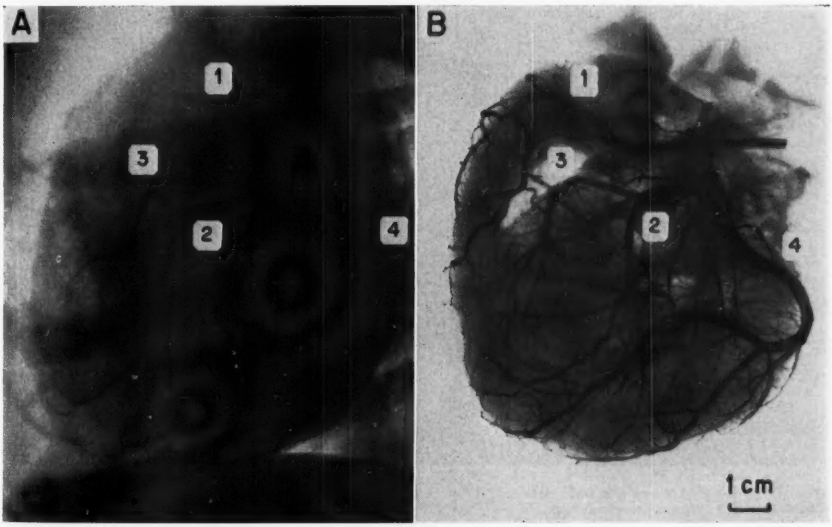

Fig. 3.—Coronary arteriograms, in vivo (*A*) and post mortem (*B*). The markers indicate the right coronary artery (*1*), and the anterior descending (*2*), septal (*3*), and circumflex (*4*) branches of the left coronary artery. The origin of the septal artery lies within the left coronary ostium. The cannula extending vertically across the heart in *A* has been cut short in *B*. A second cannula leading to the right coronary ostium is seen in *B*.

The air-filled cavity of the right ventricle shows in *B* between the right coronary and septal arteries. The arch of the aorta may be seen, containing contrast material in *A* and air in *B*.

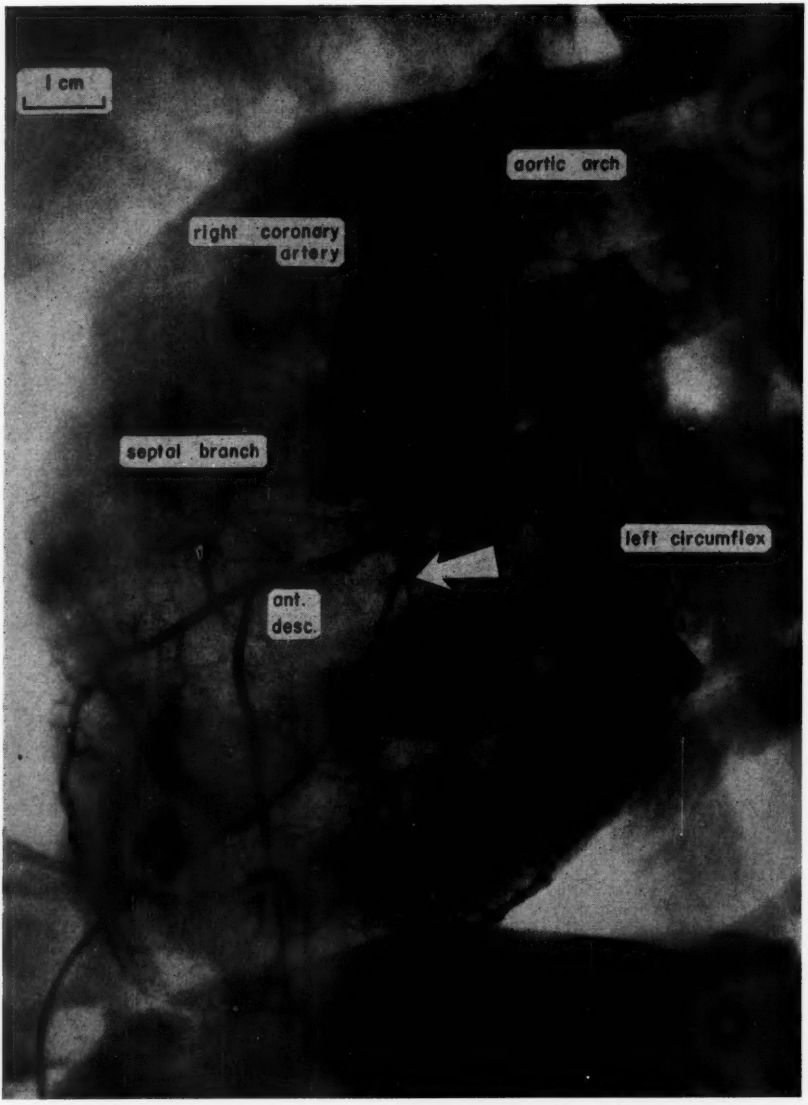

Fig. 4.—Coronary arteriogram, slightly enlarged, in a normal dog, taken by the technique described for single angiograms. The resolution is considerably greater than in the serial angiograms (Figs. 2 and 5). The heart is presented in lateral view with its left aspect toward the film. The site of insertion of the polyethylene tube at a point of subdivision of a coronary branch is marked by the arrow.

The contrast filling of the left coronary system is even. Arborizations of the third order to the ventricular myocardium can be distinguished; some atrial vessels are also seen. The right coronary artery and several of its arborizations are visible with low, somewhat uneven contrast. The cardiac veins are not outlined.

The aortic bulb is well filled with contrast material; all three sinuses of Valsalva are well outlined. The contrast is irregular in the arch of the aorta and its branches.

The contrast medium filled the proximal portion of the left coronary artery and jetted out into the aorta, sharply outlining the left sinus of Valsalva. Serial angiograms demonstrated filling of the left coronary system by the first 2 to 3 c.c. of contrast medium. Additional medium escaping into the aorta was washed away with each systolic ejection of blood from the ventricle so that, in individual exposures, the ascending aorta was frequently clear, while contrast medium and blood formed an irregular, swirling mixture in the aortic arch and in the descending aorta. The thickness of the wall of the descending aorta

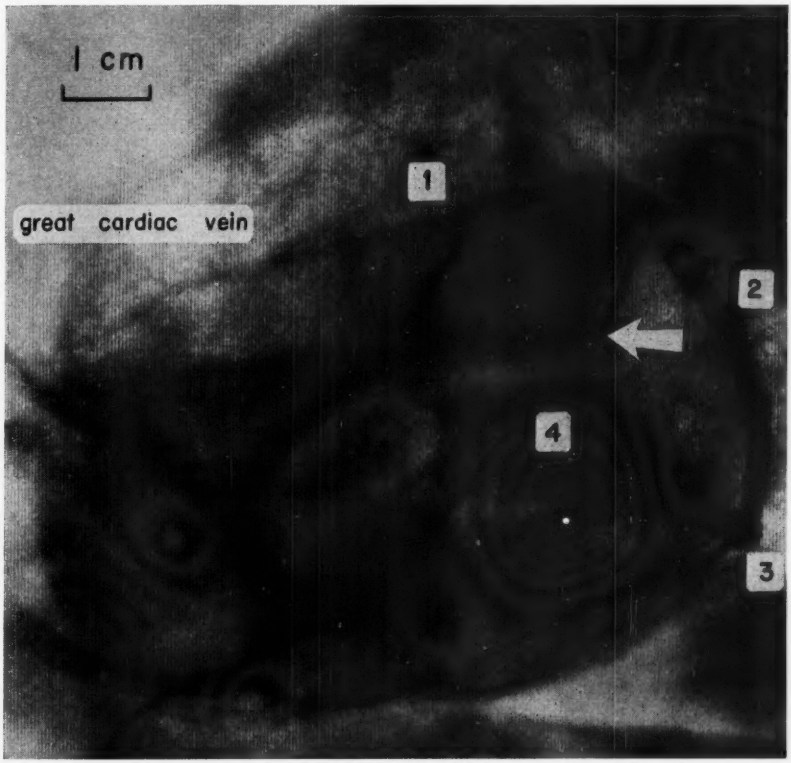

Fig. 5.—Venous phase of coronary angiogram (approximately natural size). Exposure 3 seconds after the end of the contrast injection, in a series taken with an impulse timer and cassette changer.

The polyethylene tubing still contains contrast medium but there is no remaining arterial contrast except in the small branch to the right and below the end of the tube (*arrow*). Partial occlusion by the tip of the tube apparently delayed the disappearance of contrast material from this branch.

A first order venous tributary is seen on each side of the cannulated arterial branch (no longer visible but indicated by the end of the tube). These and similar venous tributaries enter the great cardiac vein, which accompanies the anterior descending coronary artery on the anterior surface of the heart (*1*), then curves to lie transversely in the atrioventricular groove (*2*), follows the groove to the posterior aspect of the heart (*3*), and continues to the right to become the coronary sinus (*4*). The image of the coronary sinus is superimposed over that of the end of the tube. Contrast material emerging through the ostium of the coronary sinus forms a sickle-shaped shadow outlining the auricular wall.

The photographic contrast in the venous phase of the angiogram is lower than that of the arterial phase.

was shown by the width of the space between the column of contrast medium within the lumen and the negative shadow of periaortic fat and areolar tissue.

The right coronary artery was delineated to some extent in most arteriograms, but usually in discontinuous segments, in relation to the discontinuous entry of contrast medium from the root of the aorta. Even when the arborization of the right coronary artery was shown quite extensively, the photographic contrast was low (Fig. 4).

Cardiac veins were visualized in most single angiograms. Serial angiograms showed the first signs of venous delineation within 1.5 seconds of the start of the injection. Contrast filling of the veins increased during the remainder of the injection period, and persisted while the arterial contrast disappeared during the 2 or 3 seconds followed in serial radiograms after the completion of the injection. The main cardiac veins and their first and second order tributaries could be distinguished (Fig. 5), but photographic contrast was never as high as in the images of the arteries.

A faint haze due to capillary leakage often appeared in the area of dissection of the cannulated coronary branch, but this did not interfere significantly with the delineation of the vascular pattern.

When the catheter was left in situ, patency and free bleeding could be maintained without undue inconvenience for about 2 weeks. Then the last few millimeters of the tube became occluded by clot formation. Clotting did not extend into the vessel. The tube lumen could usually be cleared by the injection of saline, and the dislodged clot could then be identified in subsequent arteriograms as an embolus in the terminal distribution of the anterior descending coronary artery, unless the saline was injected sufficiently forcefully to eject the clot into the aorta. Clotting, undue embolization, or the development of leakage at the point of cannulation brought these experiments to an end. Repeated arteriograms during this period showed excellent reproducibility of the initial pattern apart from the small clots introduced. There was no evidence of thrombosis in consequence of the repeated injections of Hypaque and no visible collateral branches were stimulated by the procedure. The portion of the cannulated branch beyond the site of ligation could occasionally be seen in arteriograms taken after 1 week.

DISCUSSION

The surgical trauma involved in a direct approach to the heart for coronary angiography is no doubt greater than that of retrograde catheterization of the aorta. However, as stated at the outset, this method was developed for use in association with experimental procedures which in themselves require exposure of the heart. Suitable x-ray equipment in the experimental laboratory permits the taking of angiograms while the chest is open. Besides being convenient for decannulation, the open chest is an advantage in dealing with the occasional occurrence of ventricular fibrillation.

The introduction of contrast medium into the coronary vessels provoked slowing of the heart rate and T-wave changes, as has been observed during

coronary angiography via the aorta. It is not certain to what extent this is a chemical injury or simply due to displacement of blood. The latter mechanism is suggested by the quite similar electrocardiographic changes we produced by injecting saline. However, even if the injection of Hypaque also introduced an element of chemical trauma, the electrocardiographic changes were transitory and no evidence of lasting damage was found on histologic study. Hypaque has been left within coronary arteries by others during periods of cardiac arrest exceeding 1 hour without evidence of harm.[20]

The main hazard of coronary arteriography appears to be the risk of producing ventricular fibrillation during or just after the injection of contrast medium. Systemic anoxia due to poor respiratory exchange, and local anoxia due to prolongation of the injection period undoubtedly increase this risk. The amount of contrast material injected and the period of displacement of blood from the left coronary system can be reduced; the serial angiograms showed optimal filling and contrast by the time 2 to 3 c.c. of contrast material had been injected. For successful single exposures with a small amount of contrast medium, the x-ray unit must be triggered by a mechanism synchronizing the exposure with the brief period of contrast filling of the vessels.[8]

The ligation of the coronary branch used for injection regularly caused some myocardial damage in spite of the extensive backflow noted at cannulation. These collateral connections apparently increased in function after the ligation, because the distal portion of the branch sometimes appeared in angiograms taken a week or more after cannulation, although the collateral vessels themselves never became large enough to be seen in in vivo angiograms. The sacrifice of a branch for cannulation is readily worked into an experiment on the development of coronary collaterals, but may otherwise be a disadvantage.

What recommends the method is the high quality of the angiograms obtained, using standard radiographic equipment. Single-exposure arteriograms delineated the coronary arterial vessels almost to the limit of naked-eye resolution, i.e., to 0.2 mm. in diameter, as measured in the post-mortem injection. There was little variation in the in vivo pattern visualized in repeated angiograms during a single sitting. The physical basis of the method gives reason to expect this reproducibility. The only variation observed was in branches at the limit of in vivo visualization; this was probably a variation in definition due to cardiac and respiratory motion rather than a true change in vessel diameter. Comparison of in vivo angiograms with the more detailed studies in the excised hearts gave further confidence in the reliability of the method. The injection of contrast material in itself did not provoke any change in vascular pattern or lead to the closing or opening of vessels of the size visualized.

Arteriography by retrograde injection through a distal branch depends upon the arterial pressure of the living animal to oppose backflow of contrast medium and ensure filling of the system under study. In a nonliving animal, the contrast material would flow through the injected branch and the stem vessel and would then be dissipated into the aorta. In the living animal, the injection pressure must be sufficiently high to accomplish momentary displacement of blood by contrast medium and to prevent interruption of the contrast

column by jets of blood from the aorta. Some contrast medium is therefore lost into the aorta, but filling of all branches of the left coronary artery is ensured, including those which arise within the coronary ostium, as a large septal branch not infrequently does (see Fig. 3).

The present work was applied to the left coronary system. Some visualization of the right coronary artery was usually also obtained, but it was too fragmentary and unpredictable to be useful. However, if the major interest lies in the right coronary system, a branch of the right coronary artery can be selected for the injection.

The stereoscopic views were taken by successive exposures and did not always catch identical phases of injection, respiration, or cardiac action. These were not, therefore, true stereo-pairs in a photogrammetric sense. However, they could be viewed comfortably in a mirror stereoscope and the irregularities observed did not interfere with the identification of the vessels examined.

The method in general seems suitable for its intended purpose of delineating coronary arteries in association with experimental procedures requiring a direct approach to the heart. Its definition is sufficient, for example, to demonstrate the major vascular pattern prior to the application of a procedure designed to augment coronary collaterals. The method can provide repeated observations in vivo. It may be possible to utilize single angiograms of this kind clinically, in association with direct surgical procedures upon the coronary arteries, particularly if the branch used for injection can be saved by immediate removal of the cannula or needle or by the incorporation of that branch in the vascular reconstruction.

SUMMARY

A method is described for coronary angiography in the surgically exposed heart of the dog, using standard radiographic equipment. The method was designed to provide a detailed in vivo demonstration of the pattern of the left (or the right) coronary artery immediately prior to a direct experimental or surgical procedure upon the coronary vessels. The angiographic method was tolerated well. Coronary branches were reliably delineated down to third order divisions, corresponding to a diameter of approximately 0.2 mm.

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AORTO-ESOPHAGEAL FISTULA: A LATE COMPLICATION FOLLOWING INSERTION OF A HUFNAGEL VALVE

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THE exsanguination of a patient due to an aorto-esophageal fistula 22 months after insertion of a Hufnagel valve warrants attention being directed to this complication. Since the initial report of Hufnagel,[1] this type of prosthesis has been utilized in numerous clinics to palliate the sufferings of a patient with severe aortic insufficiency. Several complications have been recognized incident to this procedure and include septicemia, aneurysm formation at the site of aortic transection, and embolism secondary to thrombosis in proximity to the prosthesis.

The following case report describes a delayed complication not heretofore noted.

CASE REPORT

C. A., a 32-year-old white man, entered the University of Kansas Medical Center on April 23, 1954, with a heart murmur first discovered in 1938. There was no definite history of rheumatic fever.

He had been well until August, 1948, when he had an upper respiratory infection followed by recurrent episodes of weakness, fever, a productive cough, a sense of tightness in the upper chest, headache, anorexia, and night sweats. These symptoms caused his admission to the Veterans Hospital in Los Angeles in 1949.

The physical findings and his subsequent hospital course were compatible with subacute bacterial endocarditis superimposed on rheumatic heart disease. He was maintained on massive antibiotics (penicillin, streptomycin, and Aureomycin) for 13 weeks. At the time of dismissal he complained of mild dyspnea and a pressing substernal pain radiating down both arms.

He was readmitted in 1950 because of recurrent febrile episodes. From 1950 to 1952, he was relatively asymptomatic. In 1952, he had attacks of giddiness and syncope, resulting in loss of consciousness. Episodes of substernal and precordial chest pain radiating to the neck and the ulnar distribution of the left arm became more frequent. These pains were variously described as sharp, dull, or "pressure-like." They were unrelated to exertion, occurred frequently at night, and were not relieved by nitroglycerin. Ankle edema was noted during the several months previous to hospitalization in 1954.

Physical examination, when admitted to the University of Kansas Medical Center in 1954, disclosed a blood pressure of 154/40 mm. Hg. The heart had a normal sinus rhythm and was enlarged to the left with a diffuse apical impulse in the fifth interspace at the anterior axillary line. Over the aortic area a Grade II systolic thrill was palpable, and a Grade IV harsh systolic and a Grade III diastolic murmur could be heard. These murmurs were transmitted over the entire precordium and were of Grade II intensity at the apex. There was a Corrigan pulse. The lung fields were clear to percussion and auscultation. The liver was palpable 3 cm. below the right costal border and was not tender. No ankle edema was noted.

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Received for publication Jan. 17, 1958.

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Fluoroscopy showed the heart to be enlarged, and "boot shaped" with marked hypertrophy of the left ventricle, and a clearance angle of over 90 degrees. Electrocardiograms showed left ventricular hypertrophy. Other routine laboratory studies were within normal values, including tests to determine rheumatic activity.

Because of the patient's incapacity and the progress of his symptoms during the past several years a Hufnagel procedure was advised. This was done on April 30, 1954, with occlusion of the thoracic aorta for 7 minutes and 50 seconds. He tolerated the operation well and was dismissed after an uneventful recovery for further convalescence in the Veterans Administration Hospital. His only complaint was that of incisional pain, apparently distinct from the angina previously noted.

Postoperatively, a decrease in the heart size was noted (Fig. 1).

The episodes of weakness and syncope decreased markedly. Following dismissal from the Veterans Hospital the patient returned to work where he drove his own truck and managed a fruit market.

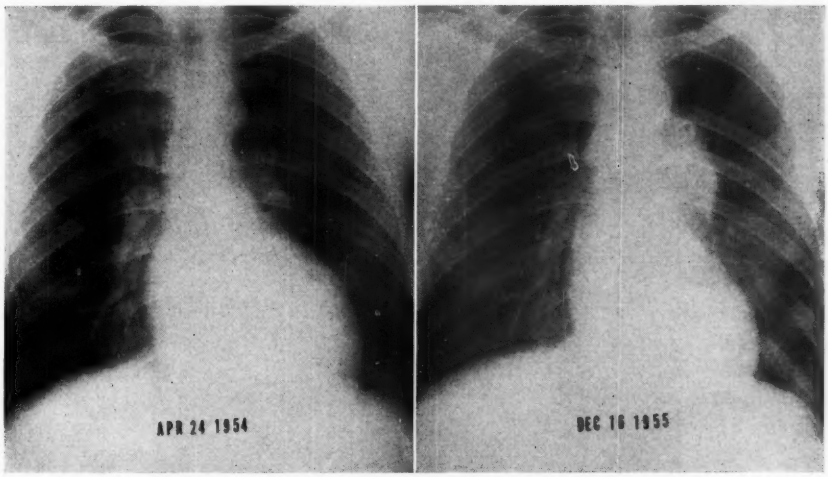

Fig. 1.—Chest roentgenograms before and after insertion of the Hufnagel valve. Note particularly the obvious reduction in heart size postoperatively and the mediastinal shadow in the area of the Hufnagel valve insertion.

The chest pain recurred, however, and was described by the patient as a feeling of pressure from the substernal area radiating down to both elbows. It was not relieved by nitroglycerin.

He was readmitted to the Kansas City Veterans Administration Hospital on Feb. 2, 1955, because of fever of 103° F. and chest pain. Blood studies disclosed a hemoglobin of 8.6 Gm. per cent and a white cell count of 15,000. He was placed on various combinations of antibiotics and Benemid which did not result in clinical improvement, and subsequently were changed to Terramycin, erythromycin, and massive doses of penicillin. During several weeks the temperature gradually decreased to normal. One blood culture was positive for hemolytic staphylococci. The previous complaint of pain persisted, but was controlled by the oral administration of codeine or Demerol. He was dismissed on May 11, 1955.

From May until December he was able to work, but was again hospitalized on December 15 with complaint of pain along the right costal border. This pain did not occur with exertion and was constant in severity and occurrence. No dyspnea accompanied it, and the discomfort did not suggest angina. It was managed by phenobarbital, Reserpine, and the occasional use of narcotics.

On Jan. 26, 1956, he was admitted to the hospital for the last time with a history of a severe pain for 5 days at the upper end of his sternum, radiating inferiorly. This had increased in frequency and severity, and was associated with dyspnea, nausea, and vomiting.

Physical examination disclosed an acutely ill patient with a temperature of 100° F., a white cell count of 11,800, and a hemoglobin of 13.6 Gm. per cent. A roentgenogram of the chest showed no changes from those noted in previous years. For the next 6 days he remained febrile and was given penicillin. Subsequently the pain in the chest increased and radiated to the mid-dorsal and lumbar areas, and to the right side of his chest. On Feb. 19, 1956, his fever increased to 103° F. and was accompanied by upper abdominal pain. During the next 10 days he received penicillin and streptomycin. The severe pain in the lower part of his chest and upper abdomen persisted, accompanied by abdominal cramping and distention. Over the course of the next 3 weeks the pain gradually disappeared and for several days he was relatively asymptomatic. On March 5, 1956, he had massive hematemesis treated by the administration of blood and dextran. A few hours after the first hemorrhage he had a second massive hematemesis and died, 22 months after insertion of the plastic aortic valve.

Fig. 2.—Photograph of gross specimen showing the aorto-esophageal fistula and the esophageal ulceration. *A*, aorta; *E*, esophagus; *HV*, Hufnagel valve.

*Post-mortem Examination.**—No free fluid was noted on opening into the pleural cavities. The pericardial sac was intimately adherent to the epicardium. At the first portion of the descending thoracic aorta a prominent mass was noted attached to the esophagus and trachea. Examination of the intestinal viscera showed them to be distended by dark blood. No other abnormalities of the abdominal viscera were noted.

The heart was removed in toto with the aorta, trachea, and the esophagus. It was grossly hypertrophied. The coronary arteries appeared normal.

Examination of the interior of the heart showed a shiny endocardium with rather marked hypertrophy of the papillary muscles and the trabeculae carneae of the left ventricle and to a lesser degree of the right ventricle. The cusps of the aortic valve were sclerotic, thickened, and calcified, although the sinuses of Valsalva and the coronary orifices were

*I am indebted to Dr. James A. Turner, Chief, Laboratory Service, Veterans Administration Hospital, Kansas City, Mo., for performing the post-mortem examination and interpreting the microscopic findings.

normal. The myocardium of the left ventricle measured 16 mm. and that of the right 4 mm. in thickness. The circumferences of the valves were: tricuspid, 14 cm.; pulmonic, 7.5 cm.; mitral, 10.5 cm.; and the aortic, 7 cm.

The aorta was dissected free from the previously described mass. Its elasticity was well preserved without visible areas of atheromatosis. The Hufnagel valve was present in the first portion of the descending aorta although it had completely lost its attachment proximally with the aorta. Its attachment with the aorta distally was intact. A thick fibrous membrane connected the proximal and distal portions of the aorta and enveloped the valve. Upon opening the esophagus, several large areas of ulceration at the level of the prosthetic valve were noted (Fig. 2). One of the ulcerations had perforated to communicate with the space between the valve and its fibrous covering. Here clotted blood and partially organized thrombi were found. Below the level of the prosthesis the aorta appeared normal.

The lungs revealed a small amount of clotted blood in the bronchi and bronchioles. The parenchyma was well aerated and elastic with a normal appearance of the major blood vessels.

Microscopic examination of the aorta disclosed a fistulous tract, partially filled by laminated thrombus, surrounded by proliferating fibroblasts. A moderate amount of atherosclerosis and adventitial inflammatory reaction was noted. Evidence of old hemorrhage was indicated by hemosiderinophages. Sections of the heart and aortic valve showed endocardial thickening, mild interstitial fibrosis of the myocardium, and fibrocalcific changes in the valve leaflets.

COMMENT

It appears most likely that this patient developed a false aneurysm at the proximal point of insertion of the Hufnagel valve. This might have occurred as a direct result of weakening of the aortic wall due to erosion of the rigid prosthesis. A second possibility is a smoldering infection in this region with abscess formation and fistulization to the esophagus. The repeated febrile bouts postoperatively suggest an inflammatory process although the microscopic evidence did not confirm this.

The benefit obtained by this patient for the early part of his postoperative course was considerable and enabled him to be employed gainfully. He also remained free from congestive heart failure.

Experiences with the Hufnagel type of prosthesis have been widely divergent. Due to the far-advanced heart disease of the patients for whom this is advised and the postoperative complications, the procedure carries a significant mortality and morbidity. The long-term results of those who survive, however, have in most instances been judged as excellent to good. As stated by Bland and Wheeler[2] in their account of 14 patients, "Thus, our experience with the artificial valve has been satisfactory in 40 per cent of a small series of 14 patients. In a few the results have been remarkable to an extent that has encouraged us and others to accept and to advise this physiologic compromise for those in serious difficulty until a more promising procedure is available."

Our own small series of 10 patients is summarized in Table I. Particular attention should be noted to patients 3, 8, 9, and 10. The first is living, well, and working daily as a farmer. The next patient successfully completed a full-term pregnancy without difficulty while the last 2 patients are relieved of their previous symptoms of congestive failure. That such an unfortunate complication as an aorto-esophageal fistula may occur 22 months after the operative procedure must be evaluated in assessing the risk of this procedure. However, until a

TABLE I

CASE NO.	INITIALS	SEX	AGE	DIAGNOSIS	DATE OF OPERATION	RESULTS
1	K. R.	M	30	Rheum. H.D.	April, 1954	Uremic preoperatively; died renal failure on 2nd postop. day
2	B. G.	M	33	Rheum. H.D.	April, 1954	Died 21st day postop. of septicemia
3	R. M.	M	62	Rheum. H.D.	May, 1954	Living and well; works daily as farmer
4	C. Z.	M	23	Rheum. H.D.	July, 1954	Died during anesthetic induction
5	R. S.	M	63	Syphilitic H.D.	July, 1954	Died 16th day postop. from mesenteric embolus
6	B. W.[3]	M	33	Marfan's D.	March, 1955	Died 4 months postop.
7	C. M.	F	44	Syphilitic H.D.	March, 1955	Good result, but died 7 months postop. from empyema of gall bladder
8	C. G.	F	19	Rheum. H.D.	April, 1956	Living and well; had full-term normal pregnancy
9	M. H.	F	50	Rheum. H.D.	Sept., 1956	Living and well
10	R. T.	M	21	Rheum. H.D.	Dec., 1956	Living and well; working daily

satisfactory method can be devised for the direct correction of aortic valvular deformity or for the insertion of a new competent aortic valve of some type, the Hufnagel prosthesis appears to offer appreciable palliation for the patient with severe aortic insufficiency.

SUMMARY

1. A 32-year-old white man with severe aortic insufficiency due to rheumatic heart disease was subjected to operation with insertion of a Hufnagel type of prosthetic valve. Appreciable objective and subjective benefit resulted postoperatively.

2. Twenty-two months postoperatively, after 5 days of acute illness, he suddenly died with massive hematemesis from an aorto-esophageal fistula at the proximal site of the prosthetic valve insertion.

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RESECTION OF CARCINOMA OF THE MID-THORACIC ESOPHAGUS WITH PRIMARY ANASTOMOSIS

A Report of 4 Cases, and One 9½-Year Cure

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IT IS common knowledge that the prognosis for carcinoma of the esophagus above the cardia approaches zero. So true is this that one well-known esophageal surgeon has stated jokingly that if he were afflicted with the disease, he would insist only on periodic dilatations to ensure the steady flow of ethanol from his pharynx to his stomach! At Johns Hopkins there has never been a 5-year survival regardless of the level of the lesion.[19] In 1954, the dean of esophageal surgery, Sweet, reported 145 cases of mid-thoracic carcinoma followed for 5 years with only 2 survivors.[18] Other reports attest to the inadequacy of surgical methods in effecting significant cures.[5, 6, 9, 10, 12, 13, 16, 17] Actually the most encouraging results have been reported by the radiologists,[1, 3, 4, 15, 17] but even with superior statistics they have demonstrated consummate reluctance in suggesting that they should therefore assume this problem.

It would be repetitious to enumerate the fundamental anatomic facts which render carcinoma of the mid-esophagus hopeless. One has only to glance at a cross section of the thorax to realize that an adequate cancer operation is impossible. The cure of the individual carcinoma of the mid-thoracic esophagus is a matter of luck. The management of the individual case is an exercise in common sense.

With this in mind we were attracted to the idea of restoring continuity by the simplest and least traumatic surgical means, primary anastomosis. Such a technique obviously limits the extent of resection and is, therefore, subject to the criticism of disregarding submucosal spread.[14] Conversely, it may be argued that those patients with submucosal extension are already beyond the curative stage and that, therefore, a palliative procedure to restore deglutition promptly is the therapy of choice.

In any event, the following descriptions represent our experience with this operation.

CASE REPORT

CASE 1.—A 56-year-old white housewife was admitted to the Harrisburg Hospital on May 18, 1948, with the chief complaint of dysphagia. She stated that for 15 months she had had progressive difficulty in swallowing solid foods, although liquids gave her no trouble. The food stuck about halfway down and was regurgitated undigested in a few minutes.

Received for publication Dec. 23, 1957.

Her general health was otherwise good and she had no significant complaints. She had a good appetite. Her past medical history revealed that in 1934 she had undergone a left simple mastectomy for chronic cystic mastitis, and a supravaginal hysterectomy for fibroids. In 1939, the right breast was amputated for cystic mastitis. The family history was noncontributory.

On May 21, 1948, esophagoscopy demonstrated a mid-esophageal bleeding friable tumor, biopsy of which was diagnosed as a Grade III squamous-cell carcinoma. The red cell count was 4.22 million per cubic millimeter, white cell count 5,700 per cubic millimeter with a normal differential count. Hemoglobin was 12.6 Gm. or 84 per cent. Urinalysis was normal, except for a trace of albumin.

On the fourth of June, a left thoractomy was performed through the periosteal bed of the resected sixth rib. The lung was not adherent. On exposing the esophagus, a hard nodular lesion approximately 2 inches long just below the region of the aortic arch was encountered. There were two adjacent lymph nodes. A primary anastomosis was effected with two rows of interrupted sutures of nylon, but this was under considerable tension. A Levin tube was passed through the nose past the anastomosis into the stomach. Fibrin foam was placed around the anastomosis and the chest was closed in layers after an intercostal catheter had been brought out through a stab wound.

The pathologic report on the surgical specimen confirmed the endoscopic biopsy report of squamous-cell carcinoma. In addition, one of the adjacent lymph nodes contained metastatic epithelioma. Grossly, the tumor was described as ulcerated, friable, and circumferential.

Postoperatively her course was complicated by the development of an esophago-pleuro-cutaneous fistula which was quite evident on the tenth postoperative day. The Levin tube which had been removed on the ninth day was re-inserted and penicillin resumed. From the twenty-fourth postoperative day, she was afebrile and was discharged 34 days after operation eating a full diet with but slight difficulty. She weighed 80 pounds.

In the 6 weeks that followed, this dysphagia progressed. Although it was necessary to re-admit her to the hospital on Aug. 26, 1948, she had gained 12 pounds. Esophagoscopy the following day revealed a granular stenosis at the operative site, but a No. 18 Fr. dilator was passed. Subsequently esophageal dilatations were performed upon her as an outpatient, but the stenosis progressed. On Nov. 22, 1948, a Witzel gastrostomy was performed under Pentothal sodium and nitrous oxide anesthesia. She gained weight but was chronically ill in spite of the fact that she could swallow soft foods without difficulty.

On Oct. 14, 1949, a left lower lobectomy was performed for a lung abcess secondary to a bronchoesophageal fistula. Her recovery was uneventful. Subsequently she gained weight, and although periodic esophageal dilatations were necessary, she had little trouble. On April 16, 1951, a No. 28 Fr. dilator was passed without difficulty. Eighteen months later routine esophagoscopy revealed no difficulty and only slight stenosis (Dec. 23, 1952).

In the ensuing years she has been asymptomatic. She eats a full diet without difficulty. Yearly esophageal checks have been normal, except for dilatation of a mild stenosis at the last esophagoscopy on Dec. 12, 1957. The last esophagogram in July, 1956, was normal except for two traction diverticula. X-ray studies of the chest were negative.

She is alive, relatively asymptomatic and well in December, 1957, 9½ years after esophageal resection with primary anastomosis.

Few authors have discussed primary anastomosis for carcinoma although it is the accepted method of restoration for children with congenital stenosis.[7, 8] In 1949, Parker and Brockington[11] reported 2 cases done for palliation; one patient lived 9 months. They demonstrated in dogs, moreover, that the technique with extensive mobilization is feasible.[11] In our patient operated upon, in 1948, anastomosis was accomplished with very little attempt at mobilization. This resulted in considerable anastomotic tension and indubitably accounted for the postoperative complications.

In October, 1956, we performed a similar operation through the right chest for a carcinoma at the level of the aortic arch. Unfortunately the patient had a metastatic mass involving the hilum of the right lung and mediastinum, which was left in situ. By mobilizing the esophagus extensively, however, we were able to make a two-layered nylon anastomosis without tension. Except for a temperature rise to 100° F. on one occasion within the first 24 hours, the recovery was completely afebrile and uneventful. The patient was eating a full diet without difficulty on the eighth postoperative day. Subsequently, esophagoscopy was performed twice and although there was moderate stenosis and granulation tissue at the anastomosis he ate a soft diet without effort. A No. 28 Fr. bougie was passed easily on May 7, 1957. Biopsies on two occasions were negative for a recurrence of the neoplasm. The patient died elsewhere in July, 1957, apparently as a result of his disease.

When this was the extent of our experience with primary anastomosis after resection, we were understandably encouraged and wondered why such a simple procedure was not the standard one. Our ardor was quickly dampened and our question soon answered by the next 2 patients who died postoperatively. One patient had a frank disruption of the anastomosis and died from mediastinitis. The other had only a small leak from the anastomosis and there was no interference of the blood supply in spite of the fact that we had mobilized the esophagus from the cervical region to the diaphragm. The cause of his death is obscure because the mediastinitis was not overwhelming. In both of these cases, the anastomoses were very difficult to complete because of the tension despite almost complete esophageal mobilization.

It is impossible to draw logical conclusions from 4 cases. We consider Cox's attitude on this subject to be both humble and intelligent. He states, "The object of the surgeon must be, therefore, to restore to the patient ease of swallowing for as long as he can contrive. If by doing a good operation in a favorable case he achieves a long-term cure so much the better, but his attention must, I submit, be focused on restoration of function as an objective of prime importance."[5] Therefore, we suggest that this method might be tried for palliation in a larger series by those who have access to more cases. We would recommend, however, that if an anastomosis without tension cannot be fashioned, another type of reconstruction, i.e., Berman tube[2] or esophagogastrectomy, be employed.

SUMMARY

To summarize, we have one patient with an apparent 9½-year cure following resection with primary anastomosis for squamous-cell carcinoma of the mid-esophagus with metastasis. She had a stormy course with complications requiring treatment for 2½ years postoperatively. The second patient, operated upon for palliation, had a completely uneventful recovery and no dysphagia until he died 9 months postoperatively. Two other patients died on the fifth and sixth postoperative days.

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TUBERCULOUS FISTULA OF THE ESOPHAGUS

Two Cases Successfully Treated by Surgery

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THE SUBJECT of tuberculosis of the esophagus has been reviewed by a number of writers.[1-6] It is an extremely rare disease which may reach the organ by mucosal, hematogenous or, most frequently, lymphatic routes. By the latter means, disease in adjacent structures such as lymph nodes, pleura, and vertebrae may spread to the esophagus. Fistulas between the esophagus and neighboring structures may occur as a result of contiguous infection. Often the occurrence of a fistula is preceded by the formation of a traction diverticulum of the esophagus. Generally the prognosis of untreated tuberculous esophagitis is poor, especially if stricture or esophagotracheal fistula exists. Antituberculous drug therapy will cure some of these patients.[7, 8] Surgery, except gastrostomy, has been employed rarely with varying results. It is the purpose of this communication to present 2 cases of tuberculous esophageal fistula which required surgery to effect a cure.

CASE REPORTS

CASE 1.—R. M., a 48-year-old white man, was admitted to Ray Brook on Jan. 27, 1955. In July, 1950, following injury to his chest he had developed a right pleural effusion. He received streptomycin for 20 days and para-aminosalicylic acid for 6 weeks, after which he terminated treatment against the advice of his physician. In March, 1952, he entered a hospital, where he stayed for about 2½ months. While in the hospital, he received streptomycin and para-aminosalicylic acid. In November, 1953, a diagnosis of tuberculous empyema was made after his admission to another institution. Treatment there included streptomycin and isoniazid, systemically, and streptomycin, penicillin, Tryptar, and Varidase, intrapleurally. On April 7, 1954, a right pleuropneumonectomy was performed. This was complicated by a bronchopleurocutaneous fistula. On Nov. 10, 1954, an 8-rib right thoracoplasty was performed. However, a pleurocutaneous fistula persisted until the patient's admission to Ray Brook. Tubercle bacilli were last identified in the sputum on June 1, 1954.

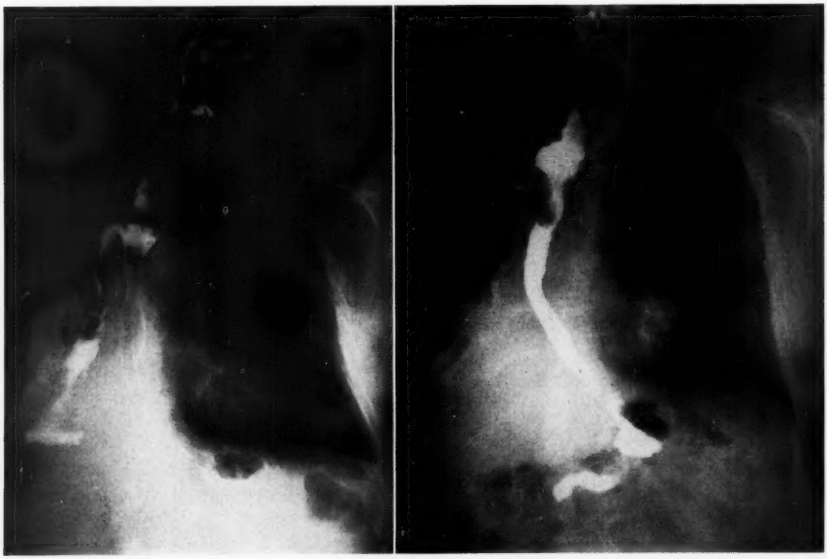
After admission to Ray Brook the patient's chest wall sinus was injected with iodized oil revealing an empyema pocket connecting with the stump of the right bronchus. No connection with the esophagus was demonstrable. Repeated cultures of sputum and empyema failed to yield tubercle bacilli. *Staphylococcus albus* and *Escherichia coli* were cultured from the empyema.

Following his admission, streptomycin 1 Gm. 3 times a week and isoniazid 300 mg. a day were administered until Feb. 7, 1957. Early in September, 1955, the patient began to experience severe coughing and choking episodes following meals. Careful questioning

From the Ray Brook State Tuberculosis Hospital, Ray Brook, N. Y.
Received for publication Feb. 3, 1958.

of the patient revealed that these symptoms had not been experienced previously. An esophagram was performed revealing a small traction diverticulum of the upper esophagus communicating by a fistula with the right pleural cavity (Fig. 1, *A*). On Oct. 18, 1955, a bronchoscopy and esophagoscopy revealed a 1 to 2 mm. dimple at the right bronchial stump and, approximately, a 4 mm. opening on the right side of the esophagus, 26 cm. from the upper incisor teeth. When the patient coughed, bubbles of air came from the opening. No other abnormalities were noted.

On Oct. 21, 1955, a modified Schede thoracoplasty was performed on the right side. The external opening of the sinus tract together with the old thoracoplasty scar were excised. The subscapular space was opened and pus and debris were removed. This space communicated with the underlying pleural space by a fistula. Regenerated ribs from the second through the eighth and a part of the ninth rib were removed subperiosteally. The thickened parietal pleura was excised preserving as many of the intercostal bundles as possible. The



A. *B.*

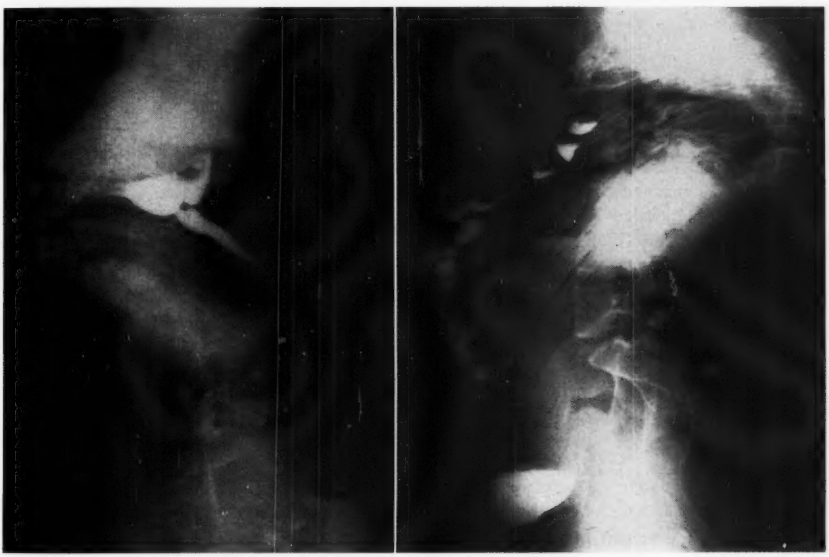
Fig. 1.—Posteroanterior chest roentgenograms after the patient had swallowed radiopaque material. *A*, Shows outline of the esophagopleurocutaneous fistula. *B*, There is no evidence of fistula following surgical treatment.

pleural cavity was then filled with isotonic saline solution and intratracheal positive pressure was developed by the anesthetist. No bronchial fistula could be identified. The stump of the right main bronchus had apparently healed spontaneously. The esophageal fistula was identified, but it was inaccessible in the apex of the pleural space. Attempts to free the esophagus were unsuccessful because of extensive scarring. Accordingly, the wound was closed in layers about a soft rubber drain. Pathologic diagnosis of tissue removed at operation was chronic granulation tissue.

On Oct. 26, 1955, a Witzel jejunostomy was performed. Following this the patient took nothing by mouth. The pleural space was allowed to heal from the depths outward. On Sept. 10, 1956, a small residual pleural pocket was obliterated by removal of several regenerated ribs with thickened parietal pleura and a partial scapulectomy. The drain was gradually shortened and the wound healed completely by January, 1957.

In January, 1956, an esophagram with iodized oil failed to reveal an esophageal fistula. This was later confirmed by a barium swallow (Fig. 1, *B*). Soon afterward the patient was able to take a regular diet without difficulty. At the end of the month the jejunostomy tube was removed. The patient was discharged from Ray Brook on May 7, 1957. In January, 1958, he reported that he had experienced no further complaints referable to his esophagus.

CASE 2.—F. M., a 26-year-old white man, was admitted to Ray Brook for the third time on Feb. 3, 1956, with fever and a tender swelling above the right iliac crest posteriorly. When he was 2 years old a kyphosis was discovered. In 1950, at 20 years, he developed coughing episodes after eating or drinking. By November, he began to experience fever. In January, 1952, a swelling appeared over the right iliac crest posteriorly. This soon opened spontaneously and drained purulent material. This together with an abscess in the right groin was drained surgically in February, 1952. As tubercle bacilli were recovered, the patient was given streptomycin and para-aminosalicylic acid therapy, which was continued until July 30, 1953. The patient was admitted to Ray Brook for the first time from March 26, 1952, to Aug. 17, 1953, because of recurrence of drainage from the iliac



A. *B.*

Fig. 2.—Lateral chest roentgenograms following ingestion of barium, showing, preoperatively, (*A*) a traction diverticulum of the upper thoracic esophagus and, postoperatively, (*B*) an outline of the substernal colon bypass. The patient has a severe dorsal kyphosis due to Pott's disease.

sinus. This healed spontaneously, and remained healed until 2 weeks prior to the patient's second admission to Ray Brook (Nov. 23, 1955, to Dec. 1, 1955), when he developed fever and tenderness at the site of the scar in the iliac region. Symptoms and signs of inflammation subsided following needle aspiration of purulent material. However, remission was only temporary, and a recurrence of symptoms soon necessitated the third admission (Feb. 3, 1956). Shortly afterward the sinus reopened, again draining purulent material.

Examination at this time revealed an acutely ill patient with a severe dorsal kyphosis. Except for a draining sinus in the right iliac region and a healed scar in the right

groin there were no relevant findings on physical examination. Repeated cultures of the sputum and pus from the sinus failed to reveal tubercle bacilli. During each of the patient's admissions to Ray Brook, esophagrams revealed a large diverticulum in the region of the kyphosis (Fig. 2, *A*). There was a delay in emptying of the diverticulum and a suggestion of a stricture below the diverticulum. Careful questioning of the patient revealed that he experienced dysphagia only occasionally when he would swallow a large bolus of food. During the third admission, a connection between the diverticulum and the sinus was demonstrated by injecting iodized oil into the latter.

During the third admission the patient experienced recurrent episodes of fever accompanied by pain in the region of the sinus tract, which continued to drain. In order to defunctionalize the esophagus, a Stamm gastrostomy was performed on Jan. 7, 1957. On Feb. 20, 1957, the cutaneous fistula was excised down to the esophageal diverticulum. The wound healed rapidly and completely from the depths outward. However, as soon as the patient was again permitted to take his feedings orally, the sinus reopened. On the other hand, as soon as gastrostomy feedings were resumed the sinus healed and remained healed as long as the patient took nothing by mouth. Since the patient did not wish to accept a permanent gastrostomy it was decided to permanently defunctionalize the esophagus by utilizing the colon as described by Mahoney and Sherman.[9] Accordingly, on June 26, 1957, the right colon and part of the transverse colon were transplanted into a substernal tunnel and, after the cervical esophagus had been severed a short distance below the cricopharyngeus muscle, the proximal esophagus was anastomosed to the cecum employing 2 layers of interrupted 4-0 silk sutures. The distal esophagus was closed by interrupted sutures of the same material and dropped back into the mediastinum. The esophagus was not severed near the cardia. The proximal end of the severed transverse colon was anastomosed to the anterior aspect of the stomach and the distal end to the proximal end of the severed ileum. Postoperatively, suction was maintained on the gastrostomy tube until gastrointestinal function returned. There were no complications. At the time of his discharge from the hospital on Sept. 2, 1957, the patient was able to take a regular diet without difficulty. Barium swallow revealed widely patent anastomoses (Fig. 2, *B*). There was no regurgitation of barium from the stomach into the defunctionalized lower esophagus when the patient was placed in approximately a 45-degree Trendelenburg position. In December, 1957, the patient reported that he had been able to return to work and had maintained his weight. He had experienced no difficulty referable to his new "esophagus" or wound in his back.

DISCUSSION

It might be argued that the fistula in the first patient was due to trauma at the time of resection of the right lung. However, the late appearance of symptoms referable to the esophagus and the presence of a traction diverticulum seem to indicate that infection was the principal etiological factor. Both traction diverticula and sinuses frequently occur in the upper thoracic esophagus as a result of adjacent tuberculous adenitis. In the second patient, Pott's disease undoubtedly preceded the formation of the esophageal diverticulum and probably was the etiological factor for this as well as the fistula. Tuberculous esophagitis secondary to Pott's disease has been reported.[4]

The role of antituberculous drug therapy in both cases was undoubtedly very important, since in both patients the tuberculous disease had become inactive by the time surgery was performed.

Excision of the fistula was unsuccessful in the second patient probably because of poor drainage of the diverticulum as a result of partial obstruction of

the esophagus below that level. On the other hand, in the absence of obstruction, healing of the fistula occurred rapidly in the first patient as soon as the empyema was successfully treated.

Combined antituberculous drug therapy is likely to render tuberculous lesions of the esophagus inactive. The lesions may heal with drug therapy alone; others require surgery to correct residual mechanical factors, such as fistula or stricture. When surgery is indicated, radical extirpation is rarely, if ever, necessary. Every effort should be made to preserve the function of the esophagus by such methods as dilatation of strictures or local resection of symptomatic diverticula. When the latter methods are not possible because of severe scarring or inaccessibility, as in the second case, a defunctionalizing operation may be used, leaving the excluded esophagus in situ. It is not believed necessary to interrupt the lower esophagus near the cardia, as was done by Johnson and associates,[10] as long as the lower esophageal occlusion mechanism is competent and can prevent regurgitation of gastric contents. Indeed, preservation of the continuity of the esophagus with the stomach permits internal drainage of the esophagus, thus obviating, at least theoretically, the possibility of infection in a totally excluded segment of esophagus.

SUMMARY

Two cases of tuberculous fistula of the esophagus treated by surgery are presented. Tuberculous esophagitis and its complications will sometimes heal with antituberculous drug therapy alone. However, surgery may be necessary to correct mechanical factors, in which cases every effort should be made to preserve as much esophageal function as possible. Occasionally, it may be necessary to create a bypass. Under these circumstances it does not seem necessary to extirpate the diseased organ.

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CONGENITAL TRACHEOESOPHAGEAL FISTULA IN THE ADULT

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CONGENITAL tracheoesophageal fistula without atresia of the esophagus is a rare anomaly. In a review of pediatric cases, published by Ware and Cross in 1954, 27 cases were reported. In the adult it is rarer still. In the 12 such cases reported in the literature,[1-11] there was a marked variation in the symptoms presented by the patient. In some, only astute questioning revealed a pertinent history.[2] In other patients, repeated respiratory infections dominated the person's existence.[3, 4, 8, 10] Other important symptoms were hemoptysis, cough productive of food, recurrent pneumonia, vomiting, and dyspnea. At times, the cough was induced by liquids but not solid food.

Radiographic studies of the esophagus using contrast media were valuable in reaching the correct diagnosis. In several instances, a gastrointestinal examination was ordered because of vomiting. The diagnosis of a tracheoesophageal fistula was a surprise finding in these cases. Radiographic study of the tracheobronchial tree was usually unrewarding. The fistula was never seen by this means if esophageal studies were normal. The bronchogram in several instances showed associated bronchiectasis.

Endoscopy was not as valuable as a gastrointestinal study with contrast media. Again, the fistula was never seen by endoscopy unless first identified by x-ray studies of the esophagus. In some cases, although the level of fistula was known, it could not be found by esophagoscopy or bronchoscopy.

The associated diseases in this small series included bronchiectasis in 3 patients[1, 9, 10]; 2 cases of mental illness—one of schizophrenia[1] and one of imbecility[4]; and 1 patient died of a carcinoma of the lung.[11] In this instance, the fistula was an incidental finding at necropsy and was situated well distant from the carcinoma. An additional patient had an associated patent ductus arteriosus and an aberrant pulmonary artery.[3]

The site of the esophageal connection to the tracheobronchial tree was reported in all patients. In 5, the communication was to the right lower lobe. Tracheal connection was noted in 4, 3 patients had openings into the left lower lobe. The fistula in the final case passed to the left main-stem bronchus.

Six cases were not treated. Three were living at the time of the report, 2 had slight symptoms,[2, 8] and 1 had moderate symptoms.[7] Three patients died with the abnormality. One woman died of asphyxia when a piece of food

Received for publication Feb. 10, 1958.

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passed through the fistula and blocked the trachea.[6] One man died of pneumonia shortly after a barium swallow.[4] The third patient died of carcinoma of the lung.

Seven patients were operated upon. Three had lobectomy and resection of the fistula (authors' case and 2 others[1, 9]). All are living and well. Two patients had pneumonectomy and excision of the tract.[3, 10] One of these succumbed to postoperative infection many months after surgery.[10] Mullard has reported another patient in whom the fistula was resected with a good result. The final patient was treated endoscopically by Clerf by silver nitrate application, with a successful result.

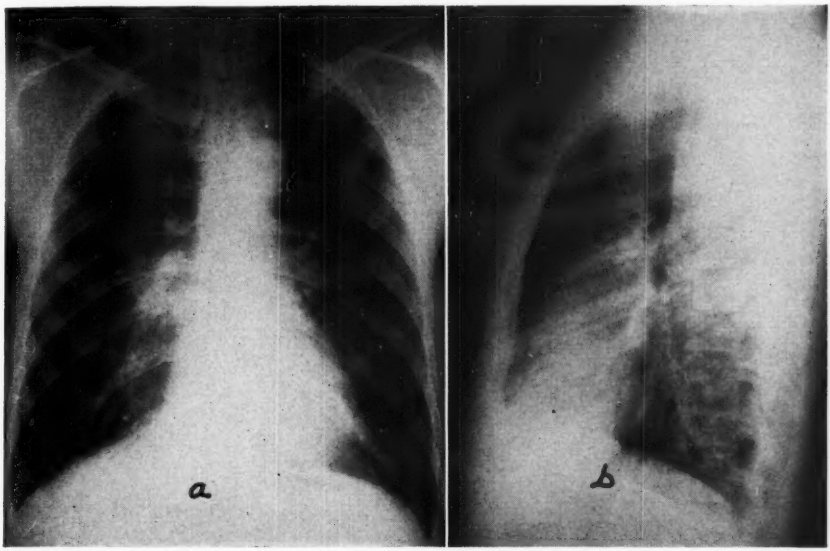

Fig. 1—Roentgenograms made on admission. *A*, Front view. *B*, Lateral view

CASE REPORT

W. A. O., a 39-year-old white man, was admitted to the Mt. Alto Veterans Hospital on July 31, 1956, because of a 2 weeks' history of hemoptysis. The hemoptysis produced dark red to brown sputum which amounted up to one fourth of a cup daily. The patient denied a previous history of hemoptysis but stated that he had had a chronic nonproductive cough dating back to at least 1945 at which time he was hospitalized for pulmonary infection. In the past 2 years, he had experienced severe coughing paroxysms on drinking hot coffee. He also recalled severe coughing spells for about the past 20 years whenever he reclined on his right side. There was no history of dyspnea, chest pain, night sweats, fever, chills, or weight loss. A history of lobar pneumonia or contact with tuberculosis was denied and the remainder of the past history was negative. The patient admitted to smoking two packs of cigarettes per day for many years.

On physical examination no significant abnormalities were noted. Blood and urine studies were normal and both sputum and gastric aspirations were negative for acid-fast bacilli. X-ray examination of the chest on Aug. 1, 1956, was reported as follows: "Chest

TABLE I. CONGENITAL TRACHEOESOPHAGEAL FISTULA IN THE ADULT

AUTHOR	AGE	SEX	SYMPTOMS	X-RAY	ENDOSCOPY	ASSOC. ANOMALY OR DISEASE	TREATMENT	LOCATION
Abrams	38	M	Hemoptysis	Fistula seen on GI series and bronchogram	None	Schizophrenia; bronchiectasis	Lobectomy and resect fistula; alive	RLL
Berglund	62	M	Cough with cold liquids	Fistula seen on GI series, not on bronchogram	None	None	None; alive	RLL
Berman	40	F	Repeated pneumonia; hemoptysis	Fistula seen on GI series, not on bronchogram	None	Patent ductus anomalous pulmonary artery	1. Close fistula; 2. pneumonectomy and resect ductus; alive	LLL
Caldwell	54	M	Repeated pneumonia; vomiting	Fistula seen on GI series; no bronchogram	None	Imbecile	None; died of aspiration pneumonia	Trachea
Clerf	23	F	Cough on swallowing for 7 yr.	Fistula seen on GI series; no bronchogram	Fistula seen at esophagoscopy		Silver nitrate applications; alive	LLL
Krausey	38	F	Vague	None	None		None; died of aspiration	Trachea
McKinney	58	F	Vomiting; cough after meals	Fistula seen on GI series; no bronchogram	Fistula seen at bronchoscopy, not at esophagoscopy		None; alive	Trachea
Moersch (Case No. 2)	52	M	Choking and dyspnea with liquids since child; repeated pneumonia	Fistula seen on GI series; no bronchogram	Fistula seen at esophagoscopy		None; alive	LLL
Morton	26	M	Chronic cough, coughs up food; several attacks of pneumonia	Fistula not seen on bronchogram; no GI series	None	Bronchiectasis	Lobectomy; resect fistula	RLL
Mullard	54	M	Purulent sputum; coughed food; repeated pneumonia	Fistula seen on GI series and bronchogram	Fistula seen at bronchoscopy, not at esophagoscopy	Bronchiectasis	Pneumonectomy; resect fistula; late death with infection	Left main bronchus
Mullard	54	F	Vomiting	Fistula seen on GI series; no bronchogram	Fistula seen with difficulty on bronchoscopy and esophagoscopy	None	Resect fistula; alive	RLL
Negus	45	M	?	None	None	Cancer of lung	None; died of cancer	Trachea
Ware and Hall	39	M	Hemoptysis; cough with liquids	Fistula not seen on bronchogram; no GI series	Fistula not seen at bronchoscopy, no esophagoscopy		Resect fistula, lobectomy; alive	RLL

J. Thoracic Surg. July, 1958

dated August 1, 1956, shows a marked prominence and increased density of the right hilum. This may be superimposition of an inflammatory process in the apex of the right lower lobe. The possibility of a neoplastic process should be considered. There are numerous blebs in the right apex. The aortic knob is prominent. The heart appears to be within normal limits" (Fig. 1). A tuberculin skin test was evaluated as 3+ while the histoplasmin skin test was negative. Bronchogram studies were reported as follows: "There is normal bronchial pattern of the right upper, middle, and all of the basilar branches of the right lower lobe. The primary branches of the apical division of the right lower lobe are also outlined. They are just above the limits of normalcy in caliber. There is no evidence of bronchial luminal obstruction or bronchiectasis." Bronchoscopic examination and right scalene node biopsy were negative. Because of the possibility of the lesion being inflammatory, it was decided to send the patient on furlough for 4 weeks and re-evaluate him after that time.

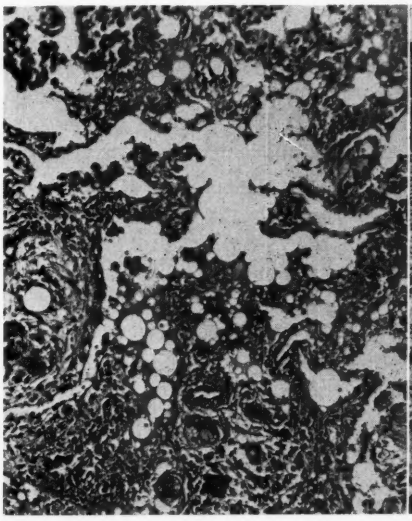

Fig. 2.

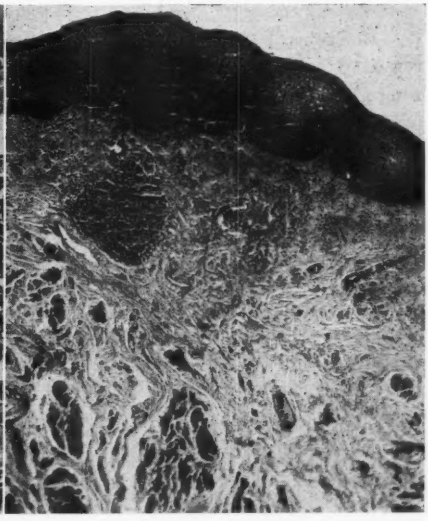

Fig. 3.

Fig. 2.—Micrograph of tissue from fistulous tract.
Fig. 3.—Micrograph which shows pulmonary parenchyma.

A chest roentgenogram taken following the furlough showed no change from the admission film. On Oct. 18, 1956, a right thoracotomy was performed. There were extensive pleural adhesions, at operation, with a mass surrounded by indurated lung tissue present in the apex of the right lower lobe. It was thought that the likelihood of tumor was sufficient to warrant right lower lobe lobectomy. After dividing the arterial and venous branches to the right lower lobe, a fistulous communication was noted between the apical segment of the right lower lobe and esophagus. The tract was resected and the lobectomy completed. A Levin tube was passed into the stomach and the esophagus was closed in two layers. Postoperatively, the patient had an uneventful recovery and on follow-up examinations is asymptomatic.

Examination of the resected specimen was reported as follows: "The right lower lobe of the lung measures 14 × 8 × 7 cm. On the upper medial surface there is a tract which on opening reveals a slightly roughened lining bronchial mucosa. This extends from the pleural surface for a distance of 2 cm. and apparently communicates with the

terminal bronchus. On bisecting the lobe, there are patchy firm areas throughout, some of which are reddish, while others are gray to yellow. [Microscopically] The pulmonary parenchyma is congested and the air spaces are filled with large hemosiderin-laden and vacuolated macrophages, mononuclear cells, edema fluid and debris. The alveolar walls are thickened and partly hyalinized. There are numerous areas of atelectasis, focal hemorrhage and several granulomatous lesions containing multinucleated giant cells of the foreign body type. Several bronchi form cystic branching spaces; there is a marked peribronchial fibrosis with accumulation of lymphocytes. [Fig. 2] Section from the fistulous tract shows a bronchial epithelium which in the peripheral sub-pleural area is replaced by squamous epithelial lining. [Fig. 3] Sections of the resected fistula (separate from lung) showed a fibromuscular tissue partially covered by squamous epithelium and described as compatible with an esophagobronchial communication."

SUMMARY

Congenital tracheoesophageal fistula in the adult is a rare entity. Thirteen reported cases are analyzed. Respiratory symptoms of hemoptysis, cough productive of food, and recurrent infection are predominant. Contrast examination of the esophagus is the best method of diagnosis. Bronchograms, bronchoscopy, and esophagoscopy are of limited value. Recommended treatment is resection of the fistula and, if necessary, adjoining lung tissue.

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CLINICAL STUDIES ON BRONCHOSPASM

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THE uncertain and frequently disappointing response to therapy of that wide variety of conditions which may be designated "bronchospastic" (e.g., asthma, bronchitis) seems to warrant a re-appraisal of the basic principles involved. The following study was conducted with this object in view.

GENERAL CONSIDERATIONS

Transverse section of bronchioles demonstrates that the muscular coat of those below 0.6 mm. diameter (i.e., at point of disappearance of cartilage and glands) is often incomplete or occasionally absent. The excellent monograph by W. S. Miller on the gross and microscopical anatomy of the lung shows that this is due to the geodetic arrangement of muscle fibers in the bronchiolar wall—the fibers being embedded in longitudinally disposed elastic tissue. In bronchioles of 0.6 mm. diameter, muscle bundles are found to vary from 0.02 to 0.03 mm. in thickness and 0.008 to 0.05 mm. in width.

The so-called respiratory bronchioles (diameter 0.25 mm.) branch terminally into two or three alveolar ducts, thence to atria and air sacs, but there are, in addition, alveoli protruding directly through the muscular latticework. It has recently been demonstrated (Lambert) that direct broncho-alveolar communications exist in the larger bronchi. These are seen bronchographically but are frequently misinterpreted as glands. The atria and air sacs have no smooth muscle in their walls.

The bronchioles are thus seen to be particularly well adapted to their function in respiration, namely, elongation and dilation alternating with shortening and narrowing. The muscular element, in view of its delicacy and relatively sparse distribution (cf., pulmonary veins and arteries of similar caliber), appears unlikely to have any appreciable independent activity. Further, bronchial elasticity is considerably less than that of the vessels as can be observed at segmental resection where the localization of subsegmental bronchi depends on this factor—that is, by traction and palpation.

Bronchi of greater caliber than 0.6 mm. are reinforced with wedges, plates, and hoops of cartilage to maintain patency. Patency beyond this would appear to depend largely on negative intrapleural pressure effecting distraction of bronchiolar walls via the contiguous aerating alveoli, and partly on the inherent rigidity of the fibroelastic coat.

Received for publication Jan. 15, 1958.

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MATERIAL FOR STUDY

Twelve young adults, 6 male, 6 female, whose ages ranged from 22 to 27 years, were studied at the operation of segmental lung resection. Two of the females and 4 of the males gave a definite history of previous bronchospastic attacks ("asthma") in childhood and adolescence, but the clinical findings in these cases did not differ significantly from the others. Apart from strictly segmental tuberculous lesions, all the patients were in good general condition, and residual lung tissue in each case completely filled the hemithorax on moderate inflation of the lungs.

The mechanism of bronchiolar respiratory movement was studied directly at operation with a hand lens. Healthy bronchioles of 0.5 mm. diameter, as measured with a stainless steel gauge, ruptured across the intersegemental plane during stripping of the segment and others, which had been stripped along their length, showed rhythmic variation in caliber, with pressure changes in the anesthetic apparatus. The effect of temporary occlusion of the "feeder" bronchus was then observed. The subsegmental bronchus to the area in which lay the bronchioles under study was occluded with a rubber-shod clamp. Respiratory excursions persisted, although more slowly, due to air drift from adjacent unoccluded segments which fllled the alveoli around the bronchioles and distracted their walls circumferentially.

On each occasion, a few of these bronchioles were removed and submitted to histologic examination. The degree of shrinkage was estimated at approximately 40 per cent. Several observers, for example, Miller and Boyd, describe a relative increase of smooth muscle at the junction of atria with respiratory bronchioles. They have even been termed "sphincters" in some anatomy textbooks (Gray and Cunningham). This appearance was found due to more marked shrinking and retraction of the bronchiole at the junction with the nonmuscularized atria and alveoli. Even so, the proportion of muscle ring thickness at this site to total bronchiolar lumen is 0.02 or 0.01 mm. to 0.25 mm. which implies that complete occlusion by mucular action is unlikely. There was no evidence of myohypertrophy in the "asthmatic" as compared with the normal individuals.

"HUMORAL" EFFECTS

The direct effect of drugs on the exposed bronchioles was observed. A few minims of 50 per cent histamine were dropped on the open bronchiole; in 2 to 4 minutes the lumen diminished. It seemed that the edema affected the mucosa particularly—there was certainly no contraction of the bronchiole which was then lightly swabbed and 0.01 per cent Adrenalin applied. Again there was no detectable change in total thickness but the lumen became wider within 10 seconds.

On seven occasions following the application of histamine, papaverine was applied instead of Adrenalin. Although Parkes found this a powerful bronchial spasmolytic, no change was observed even 5 minutes afterwards. Atropine, likewise, produced no change.

NEURAL EFFECTS

Although a fairly rich supply of sympathetic and parasympathetic nerve fibers accompanies the bronchioles, that they exercise any appreciable effect upon their caliber is far from proved. Many of these fibers are vasomotor to bronchial arteries, secretomotor to glands and afferents from stretch receptors and epithelium. Only a small proportion of them is myomotor (Mitchell).

Abbott and his co-workers in their consideration of the surgical management of emphysema and intractable asthma by denervation of the lung—a full description of the operative procedure is given—claim that postoperative bronchoscopy shows comparative relaxation of the ipsilateral bronchi and, deductively, of the bronchioles. They are less certain, however, that this is maintained and now postulate "trigger areas" of disease, such as bronchiectasis, and that the bronchospastic attacks are due to "reactivation of an inflammatory component in a localised area of destructive pulmonary disease." More significantly, they state that "in such cases resection of the destroyed area alone may produce cure" (i.e., sputum reduction).

Klassen and his associates describe a series of 7 cases in which unilateral vagotomy was performed in inoperable bronchogenic carcinoma to allay or diminish troublesome cough. Bronchoscopic and bronchographic examination (the latter in full expiratory and inspiratory phases) showed no alteration in caliber or movement of the ipsilateral as compared with the contralateral bronchi.

Autonomic innervation of the lungs is, in part at least, bilateral (Phillips), but it seems reasonable to expect appreciable visible alteration bronchoscopically following the operative procedures, described by Klassen and Abbott, which should certainly interrupt the pathways behind the main carina as, or shortly after, they traverse the midline, if neural control is major factor.

This aspect was investigated by injection of 2 per cent Xylocaine at operation in the 8 right-sided cases, since the production of even a temporary recurrent nerve palsy was considered unjustifiable in view of its adverse effect upon expectoration.

The hilar structures were exposed routinely at operation and, following removal of the affected segments, the right vagus nerve was infiltrated with Xylocaine from the level of the first dorsal vertebra to the hilum. The stellate ganglion and dorsal ganglia of the sympathetic chain were also infiltrated as far as the level of the seventh dorsal vertebra. A ring of edema was raised in the alveolar tissue around the main bronchus, main pulmonary artery, and the upper and lower veins. Some 10 c.c. of Xylocaine were employed on each occasion.

No alteration in caliber of bronchioles on the raw intersegmental surface was observed when vagus and sympathetic nerves were blocked separately and together. Immediate postoperative bronchoscopy after thoracotomy closure (the ipsilateral pupil still showing dilation) revealed no alteration of

rhythmic bronchial respiratory variation as compared with the other side of the bronchial tree. Full expansion on the side of operation was ascertained by auscultation.

DISCUSSION

Bronchospasm.—Most physiologic studies on bronchial musculature have been made on isolated bronchial chains (Hawkins and Schild) and the response of these to various drugs. The fact that such experiments ignore the powerful distraction of the bronchioles effected through contiguous alveoli by thoracic skeletal musculature would account for the consequent failure to substantiate them clinically. The experimental use of guinea pig lung which contains proportionately much more smooth muscle than human lung has further confused the issue.

Bronchoscopic examination of patients with extremely rapid respiratory rate shows bronchial rhythmicity in complete accord with thoracic and diaphragmatic excursions. In postoperative bronchoscopy of Semb-type thoracoplasty, the segmental bronchi of the upper lobe on the side of operation also exhibit "paradoxical" movement.

The relative immobility of the bronchial tree in unilateral "frozen chest" is a further example of the passive nature of the bronchial rhythm.

It seems unlikely that there is synchronous neural control of the bronchial tree during respiration. Against such reciprocal innervation of smooth and voluntary muscle is the slow rate of contraction and relaxation of which the former is capable.

The degree of ischemia and engorgement, respectively, of the lung fields during the Valsalva maneuver and inspiration against resistance (occlusion of nose and mouth) following complete expiration, can be demonstrated radiologically. The ease with which such passive alterations in the more muscular pulmonary vascular tree are induced further suggests the improbability of "spastic" bronchioles being able to maintain effective resistance.

Bronchospasm has never been reported in a patient with a dry bronchial tree. There is always increased bronchial secretion and it is unquestionable that considerable bronchiolar edema is also present. The degree of relaxation afforded in expiration, or of compression in forced expiration, will be sufficient to occlude the lumen completely.

The mechanism of bronchospasm is thus more likely to be valvular occlusion of bronchioles by edematous or thickened mucosa and/or intraluminar debris. In the few reported cases of death during asthmatic attacks (Oswald and Reid), edema and congestion of the bronchiolar wall, and particularly of the epithelium with occlusion by mucous plugs and deciduous epithelium, were marked features at autopsy.

Muscular hypertrophy has never been demonstrated in such cases and this tissue in common with the other constituents of the bronchiolar wall merely shows edema.

In allergic asthma, this concept would seem to be supported by the fact that other allergic manifestations such as rhinitis, conjunctivitis, urticaria, among others, produce maximal effect upon epithelial surfaces. The inhalation of irritating particles of dust, pollen, or gases, such as tear gas or chlorine, produces a marked increase in bronchial secretion with congestion of the respiratory epithelium. Whether the edema and congestion is due to direct irritation or to a topical antigen-antibody response, as in allergic asthma, is presently irrelevant, it seems probable that the result is identical in each instance and that muscle spasm plays no part in it.

The typical "bronchospastic" attack responds, although by no means invariably, to Adrenalin ("adrenalin resistance" as described by Carr), when administered as an aerosol spray or by subcutaneous injection, and the former method of administration is as, if not more, effective than the latter. The action is probably a vasomotor one on the bronchial mucosa. The tardier beneficial effect of atropine due to inhibition of bronchial secretion has widespread application in the postoperative management of thoracic surgical patients with troublesome copious watery sputum, but there is no experimental or clinical evidence of marked bronchodilator activity—in fact this drug has been shown to have a bronchoconstrictor action (Burstein and Parrott).

Bronchiectasis.—Bronchographic study in asthmatics is notoriously difficult but Overholt and co-workers, in a series of 76 cases of asthma (the bronchitis-emphysema syndrome), found bronchiectasis in 27, of which 13 cases were bilateral. All of them warranted resection.

As the myelastic coat of the bronchial tree seems designed primarily for the maintenance of "tone" during respiration, destruction or weakening by an infective process—either pyogenic or even tuberculous—of the bronchial walls will inevitably lead to dilation, i.e., bronchiectasis.

Three main factors are held to be concerned in the production of saccular or cylindrical bronchial dilation (Cappel).

1. Atelectasis of associated parenchyma with resultant increased negative pressure on the bronchial walls.
2. Weakening and excavation of the bronchial walls due to infection.
3. Contraction of peribronchial fibrosis.

Whether or not the surrounding parenchyma is atelectatic, consolidated, more or less normally aerated, or even emphysematous, will affect only the rate of onset of bronchiectasis for, in each instance, the bronchi are relatively wide (or in atelectatic or consolidated lungs—the only) air-filled spaces with distensible walls. Thus, in the atelectatic lung, the full distractive force is acting upon the bronchial walls alone. Since long-continued pressure changes can only be withstood by muscle tissue (cf., ligaments of joints in paralyzed limbs), when this is destroyed gradual stretching of the elastica and supervening fibrous tissue results.

Histologically, the most striking feature in bronchiectasis is the destruction of the muscular and elastic tissue of the bronchial walls with dilation, infective hyperplasia and ulceration of the bronchial epithelium. In many instances, the resected lobe or segment may show no diminution in volume and

the parenchyma both macroscopically and even microscopically appears normal. Indeed, it is sometimes a matter of some difficulty at operation to decide whether or not the appearance at thoracotomy justifies resection at all, although at the hilar dissection in such instances the extremely thin, friable state of the affected bronchi often leaves little doubt.

In a somewhat smaller number of cases, the bronchographic appearances may return to normal following conservative therapy (breathing exercises and postural coughing). In these cases of reversible bronchiectasis (D'Abreu), it would seem that the muscle has either not been severely damaged or has recovered sufficiently to exercise adequate tonal control. There are grounds for considering the possibility of smooth muscle regeneration following control of the destructive process (Maximow and Bloom), although it is doubtful if the subsequent architectural distortion would allow of complete reversal to normal.

CONCLUSION

The effect of bronchiolar muscle spasm is negligible in view of the relatively sparse distribution of this tissue. The skeletal musculature of the thoracic cage is responsible for variation in caliber. The function of the musculature of the bronchial tree is maintenance of tone. When this is lost due to damage or destruction of the myelastica, bronchiectasis occurs.

Were it possible for the so-called sphincters at the bronchiolo-atrial junction to effect closure, the sequence would be rapid absorption of alveolar air, namely, atelectasis; the alveoli, however, in severe asthma are maximally distended and emphysematous.

Present trends in therapy of so-called bronchospastic conditions suggest that undue attention has been directed toward relaxation of the bronchial musculature for, although many excellent spasmolytics are available, their effect thereon is at best variable. Antibiotics to allay infection, bronchial "detergents" incorporating trypsin and hyalase which reduce secretion viscosity and liquefy fibrinous exudates, antibody-antigen block by desensitization, and antihistamines to minimize the local edema of the allergic response are considerably more effective, although the last named are probably less so than the others.

SUMMARY

The humoral and neural influences upon bronchiolar caliber have been studied in relation to the histoanatomical structure, both directly at operation and indirectly at bronchoscopy. It is suggested that the only important factor is alteration in pleural pressure and that the mechanism of clinical bronchospasm bears little relation to intrinsic bronchial musculature, but is entirely due to edema or mechanical block of the lumina. It is further suggested that the main etiological factor in the production of bronchiectasis is loss of or damage to bronchial musculature.

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THE BRONCHUS STUMP

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Long after the general adoption of the Listerian principles in surgery had lessened the role of infection in operations elsewhere, postoperative infection remained a serious deterrent to chest surgery. Controlled, positive-pressure endotracheal anesthesia removed the fear of the consequences of the open chest. The appreciation of the importance of individual hilar dissection and the more liberal use of blood transfusions removed the fear of uncontrolled hemorrhage and operative shock. But, regardless of how the bronchus was closed, it practically always reopened in the postoperative period with the development of an empyema and a bronchopleural fistula. Thus, it was that in 1935 a mortality rate of 64 per cent in lobectomy for bronchiectasis was considered encouraging. The majority of the deaths and complications were the result of breakdown of the bronchus stump. Even today the commonest cause of failure in the surgery of lung resection, especially in pulmonary tuberculosis, is bronchopleural fistula (Table I). Because of this the author felt that it would be valuable to re-emphasize some of the principles of bronchus closure.

Table I. The Bronchopleural Fistula Rate in Pulmonary Resection for Tuberculosis Collected From Publications Between 1951-1958 From Canada, United States, Great Britain, and France

Number of Resections	Number of Bronchopleural Fistulas	Per Cent B-P Fistulas
7,750	520	6.7

CAUSES OF BRONCHUS BREAKDOWN

Infection.—Without a doubt, infection of the sutured bronchus stump is the commonest cause of its failure to heal. The possible sources of infection are extrabronchial, endobronchial, and intrabronchial.

First, extrabronchial infection may arise from accidental contamination of the pleural space from spillage of secretions during resection, from rupture into cavities, abscesses or solid (usually tuberculous) lesions during mobilization of the lung, and from breakage into or incision of infected hilar lymph nodes during dissection of the bronchus stump. Peribronchial infection, too,

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Received for publication Jan. 21, 1958.
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may result from transgression of infected tissue planes, particularly in segmental resection for pulmonary tuberculosis. Infection may also occur outside of the stump as a result of leakage of intrabronchial secretions if closure of the bronchus stump has been inadequate. A further source of extrabronchial infection is failure to eliminate rapidly the pleural dead space following resection of a segment or lobe. The results of such infection are peribronchial abscesses, with or without an overt thoracic empyema, and stump breakdown.

Second, endobronchial infection is usually a complication of the surgical resection of tuberculous lung. It has been clearly shown that tuberculous involvement of the mucosa and submucosa, at the line of section, is present in more than two thirds of resected bronchi. It depends on the degree and activity of this tuberculous process how serious a deterrent such involvement is to wound healing. Endobronchial infection is of lesser importance in the surgery of bronchiectasis or other pulmonary infections.

Third, intrabronchial infection is almost entirely a complication of poor surgical technique by which a long bronchial stump is left as a cul-de-sac for the collection and infection of retained bronchial secretions.

By any of the above modes of infection, delayed healing or actual disruption of the bronchus stump may occur since infected structures rarely heal well. A thoracic empyema may precede or follow the breakdown of the bronchus so that bronchopleural fistula and empyema are usually concomitant complications.

Devitalization.—Close to infection is devitalization of the cut end of the bronchus as a cause of stump breakdown. The bronchus heals at its end and if this end is deprived of an adequate blood supply then necrosis is inevitable.

Coverage.—Failure to cover the bronchial suture line with living intrathoracic tissue is probably one of the gravest errors in bronchus suture. The classic experiments of Rienhoff and co-workers[3] have shown that a pleural flap alone, without bronchus suture, will prevent fistula formation in the experimental animal.

Technique.—Errors in technique rank high among the causes of bronchopleural fistula. Although these will be discussed in detail later, the more important errors are too long a stump, devitalization of the stump, too much suture material, and inadequate coverage of the suture line.

PREVENTION OF BRONCHUS BREAKDOWN

With the knowledge that most bronchopleural fistulas result from infection, devitalization of the stump, or technical error, some observations can be made on their prevention.

First, no patient should be operated upon until the infection from which he is suffering is adequately controlled. This is of especial importance in pulmonary tuberculosis where endobronchial disease is such a major source of fistula formation. Months of preoperative antimicrobial therapy may be necessary before the "target point" is reached. In bronchiectasis and pulmonary

abscess, preoperative physiotherapy and the appropriate antibiotics must be administered until the bronchial tree is as "dry" and infection free as possible. These measures will ensure a healthy bronchus stump and a minimum of secretions in case of accidental spillage during resection. These points are of lesser importance in the surgery of pulmonary cancer except when the lung distal to the growth is infected.

Second, when lung overlying cavities, abscesses, or solid caseous lesions is being mobilized it must be done extrapleurally so that the lesion is not broken into and the pleural space contaminated. Except in pulmonary cancer, overzealous gland dissection must be avoided lest caseous and/or infected nodes be broken into with resulting peribronchial infection.

Third, transgression of infected tissue planes must be avoided by substituting a lobectomy for a segmental resection if the latter procedure is going to mean stripping through infected lung. This problem is almost exclusive to segmental resection in pulmonary tuberculosis.

Fourth, rapid elimination of the pleural dead space after resection is important in reducing postoperative empyema and fistula. This is obtained by careful preoperative physiotherapy and sustained postoperative physiotherapy to aid rapid elimination of the dead space. Adequate tube drainage of the pleural space with or without suction will encourage expansion of the lung. Finally if there is obviously going to be considerable discrepancy between the remaining lung and the pleural space, space reduction by phrenic crush, pneumoperitoneum, or thoracoplasty must be done.

Fifth, the most important source of breakdown of the bronchus stump is failure to observe high division of the bronchus. If the bronchus is not divided almost flush with the trachea during pneumonectomy or as close to the parent bronchus as possible during segmental resection or lobectomy, then a sump for the accumulation of secretions is left. This is the greatest source of postoperative stump infection, delayed healing, and breakdown.

Sixth, devascularization and sloughing of the stump can be limited by avoiding overzealous peribronchial gland dissection except in pulmonary cancer. Extensive peribronchial dissection will unavoidably damage bronchial arteries and lead to devitalization of the termination of the bronchus. When a bronchial artery must be controlled, a terminal transfixion stitch of fine silk is used just proximal to the level of bronchus section. Also proximal clamps are best avoided unless they are of the noncrushing type, e.g., Pott's artery clamps.

Seventh, no resection is complete unless the suture line is covered, preferably by living intrathoracic tissues. The best and simplest tissue is a pedicled pleural flap but a free pleural graft, mediastinal tissues, pericardial flaps, and even pedicled intercostal muscle bundles may be used. It has been shown that this step is more important than any other except flush resection of the bronchus stump. The animal experiments of Rienhoff[3] demonstrated this point beautifully.

Eighth, although many authors place great reliance on a particular type of suture material, the amount of suture material and the carefulness of its placement are probably as important as the type of material. It is generally agreed that nonabsorbable suture material is best, although Björk[1] recently claims equally good results with catgut. Most authors favor fine atraumatic silk or atraumatic stainless steel wire and the fewest sutures compatible with closure should be used. These must be tied firmly enough to ensure an air-tight closure but not so tight as to cause necrosis or to cut through—approximation without strangulation.

Finally, the technique of closure itself may be the cause of bronchopleural fistula. Most of the possible errors in technique have been mentioned above but these will be re-emphasized as I describe the simple technique of bronchus closure I have used in over 200 consecutive resections.

TECHNIQUE OF BRONCHUS SUTURE

Recognizing the points already discussed, the technique of closure of the resected bronchus rests upon several very important principles. These could be summarized in the following adjectives—gentleness, brevity, simplicity, and coverage. Since resection of the right upper lobe is one of the commonest resections in pulmonary tuberculosis and one of the commonest sources of fistula let us consider the surgical technique of dealing with this bronchus as our example. Let us assume that all the essential steps prior to the actual bronchus section have been performed.

Minimal peribronchial dissection is done with no stripping of tissues and no overzealous gland dissection except in cancer surgery. Thus, the bronchial blood supply is not compromised. The bronchial arteries, usually three in number, are underrun with 3-0 atraumatic silk and tied as close to the parent bronchus as possible, since this will be immediately proximal to the line of section. This ensures an adequate blood supply to the cut end of the bronchus and also prevents these sometimes formidable vessels from spurting during the closure of the bronchus—an important point if the open-bronchus technique is being used. A Brock (toothed) bronchus clamp is placed as distal as possible on the bronchus and closed completely. This prevents spill from the lung when the bronchus is cut and I have never known this clamp to come off. Then a curved Pott's aorta clamp is placed across the right upper lobe bronchus, flush with the main-stem bronchus. This is closed one notch and the bronchus is cut off about 0.5 cm. distal to this clamp which by its nature can do no harm. I have used this proximal clamp on more than 200 occasions with no complication. Its presence prevents escape of anesthetic gases and spillage of secretions, blood, or other elements into the bronchus. It also relieves one of any hurry in suturing the bronchus which can be sutured, in full view, carefully and unhurriedly after the lobe (lung or segment) has been removed.

Nonabsorbable suture material is used—2-0 atraumatic silk or No. 36 stainless steel wire on atraumatic Sellors needles. Whether silk or wire is used, two 2-0 silk stay sutures are placed through the upper and lower ends

of the bronchus. The Pott's clamp is removed and the bronchus is closed by the open technique. A through-and-through stitch is placed centrally and two additional stitches are placed on each side of this central one. The two stay sutures are tied and the ends left long. Seldom are more stitches needed to ensure adequate closure. The bronchus stump is covered by 1/1000 proflavine solution while the anesthetist applies 25 to 30 mm. Hg pressure to the suture line by squeezing on the bag to test the airtightness of the closure. As few stitches as possible are used. Two for a segment and five to seven for a lobe or lung are usual. There is no need for complex closures, curvilinear incisions, dissection or cutting of cartilages, or mattress stitches. Brevity of stump length and brevity of suture material is combined with a simple stitch to ensure firm apposition without strangulation.

A large pedicled pleural flap is then raised from the posterior chest wall and sewn down onto the suture line by means of the two stay sutures which are rethreaded. If silk has been used throughout, additional stitches are left long and rethreaded so that the pleural flap can be closely approximated to the suture line and not just "tented" over it. "Tenting" leads to the accumulation of blood around the suture line with the danger of a peribronchial hematoma forming and leading to a peribronchial abscess. Careful reinforcement of the suture line is next to high division of the bronchus in preventing fistula. The two points are complementary because a short stump (as well as being more easily covered) retracts into the mediastinum and so buries itself. Surrounding mediastinal tissues, pericardial fat, or flaps, free pleural grafts, and hinged intercostal muscle bundles can also be used but pleura is simple and adequate and generally available. Even a free pleural graft adds greatly to the tensile strength of the bronchus closure.[2]

The pleural space is washed out with proflavine and, after this is sucked out, 1 gram of streptomycin crystals and 50,000 units of bacitracin powder are placed over the site of the bronchus stump. The chest is closed leaving one apicobasal No. 40 Bardex whistle-tip catheter in the chest for the removal of air and blood during the postoperative period. This is connected to 3-bottle suction at 20 mm. Hg suction pressure.

Using the above technique, over 200 consecutive resections have been performed with only 2 bronchopleural fistulas. These 2 fistulas developed in 150 resections for pulmonary tuberculosis. Both bronchi (a right upper lobectomy and a left apicoposterior segmental resection) were closed with silk. No fistulas have occurred when wire was used to close the stump and there have been no fistulas after resection for bronchogenic carcinoma or bronchiectasis, or after segmental resection for simple pulmonary tumors or other lesions. Wire imparts no greater tensile strength to the bronchus closure[2] but, by its inertness and its nature, which requires greater care in its use, wire is the preferable nonabsorbable suture for bronchus closure. This is particularly so in tuberculosis where there is a high (histologic) incidence of endobronchitis at the suture line and in bronchogenic carcinoma where an extensive hilar gland dissection usually accompanies the resection.

SUMMARY AND CONCLUSIONS

Problems associated with the closure of the bronchus stump have been reviewed. Infection, devitalization, and errors in technique are the commonest causes of bronchus breakdown. These can be avoided by observing "flush" section of the bronchus to avoid a sump, by gentleness in peribronchial dissection to avoid devascularization of the stump, and by the use of as few sutures as possible to close the stump which must be carefully covered by living intrathoracic tissues. Wire is the preferable suture material for bronchus suture. An incidence of less than 1 per cent bronchopleural fistula is recorded by observing these principles in over 200 pulmonary resections.

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PRIMARY REPAIR OF FRACTURE OF THE LEFT MAIN-STEM BRONCHUS

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Fred E. Stull, Jr., Major, USAF (MC), Wiesbaden, Germany

FRACTURE of a main-stem bronchus is said to be a relatively common complication of severe trauma to the thorax. However, the opportunity to treat such an injury of the left main-stem bronchus is relatively rare since the patient does not usually survive because of laceration of the closely related aorta.

The condition was described in 1906 by Barford,[1] in England, at autopsy following severe chest trauma. Sanger,[2] in 1945, reported 2 cases in which successful suturing of the bronchus was carried out, but it was not until 1948 that Griffith[3] reported the first successful resection of a stricture and re-establishment of continuity 8 months following injury. The longest interval between injury and repair was 15 years. Mahaffey[4] reported a case successfully repaired 11 years following injury. The ventilatory function improved progressively in this case; however, oxygen consumption did not improve equally and the patient was partially desaturated following breathing 100 per cent oxygen.

CASE REPORT

A 22-year-old airman was admitted to the 7100th USAF Hospital on Oct. 10, 1956, following an automobile accident 24 hours previously. He was driving and had struck his chest against the steering wheel after hitting a tree.

On admission he was lethargic and breathing heavily. There was subcutaneous emphysema over the left chest with the cardiac border shifted to the right. Hamman's sign was present. There were no localizing neurological signs. Blood pressure was 108/70 mm. Hg; pulse, 96, and respirations, 28. There were decreased breath sounds over the left chest and swelling of the right supraclavicular area. Abrasions over the forehead were present.

On admission, the hematocrit was 43 per cent; the white cell count and differential were normal; urinalysis was negative. Roentgenograms revealed 60 per cent pneumothorax on the left side with severe mediastinal and subcutaneous emphysema (Fig. 1).

On the day of admission a multiperforated No. 28 colon tube was inserted into the left chest laterally and threaded up to the apex of the chest. There was egress of air and fluid revealing a tension pneumothorax. On the following day, the lung was expanded (Fig. 2). On October 12, the patient was cyanotic and dyspneic, with a fever of 102° F. An electrocardiogram was negative. Roentgenograms revealed complete atelectasis of the left lung, with marked shift of the mediastinum to the left (Fig. 3). Minimal pleural reaction was present. Bronchoscopy revealed a necrotic area completely stenosing the left main-stem bronchus, 3 to 4 cm. from the carina tracheae. The bronchoscope could not be passed beyond this area and blind

From the 7100th USAF Hospital, Wiesbaden, Germany.
Received for publication Jan. 13, 1958.

probing did not seem advisable because of the possibility of perforation into the mediastinum. A tracheostomy was performed. On October 16, bronchoscopy yielded identical findings. During this time the left lower lobe was completely atelectatic and pneumonitis developed in the right lower lobe. The patient was cyanotic even in an oxygen tent. He was treated with antibiotics and began to improve clinically as the pneumonia in the right lower lobe resolved (Fig. 4). On October 17, a planigram showed an obstructed left bronchus with a small amount of air in the bronchi beyond (Fig. 5). A bronchogram on October 19 demonstrated a 2 cm. segment of obstruction which permitted a very small amount of Dionosil to pass along the inferior border of the bronchus; this was interpreted as a fracture with granulation tissue (Fig. 6). Pleural reaction increased rapidly after October 16, to produce a massive effusion with shift of the mediastinum to the right which required repeated thoracentesis from the sixteenth to the thirtieth of October (Fig. 7).

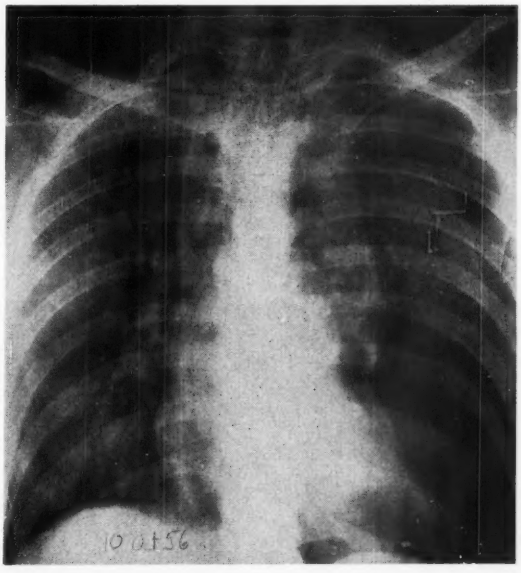
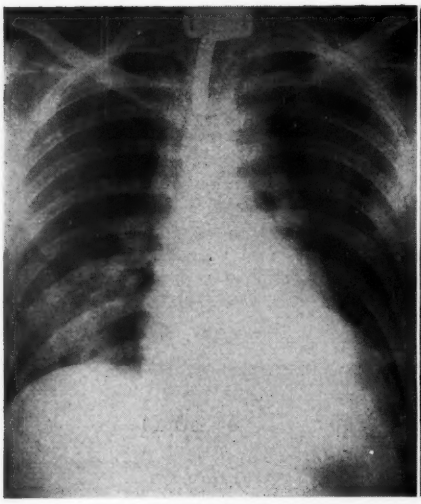


Fig. 1.—Oct. 10, 1956. The initial film demonstrates left pneumothorax, mediastinal and subcutaneous emphysema of marked degree. There is no mediastinal shift.

It was thought that if drainage of the left lung could not be established soon, pulmonary function and even the lung itself would be lost due to bronchiectasis or lung abcess; further, that the fractured superior border of the left main bronchus would continue as the source of mediastinitis and effusion. On Nov. 1, 1956, a left thoracotomy was performed. The left lung was collapsed and approximately 3 L. of fluid were present. The dissection at the left hilum was difficult because of the previous mediastinitis. After the arch of the aorta had been partially mobilized, the left main-stem bronchus was visualized and revealed a complete fracture of the bronchus which included the mucous membrane. The surrounding mediastinal structures had fallen into the fracture line causing occlusion of the bronchus. The bronchus was opened and the excess mediastinal tissue was cut away. Thick mucoid material was aspirated from the bronchus. The bronchus was closed, using one layer of interrupted No. 3-0 silk. The bronchus was not as firm on the cartilaginous side as the operator wished due to softening of two cartilaginous rings. The endotracheal catheter, which had previously been down the right main-stem bronchus during the operation to isolate the left side, was moved back into the trachea and the left lung expanded and deflated well.



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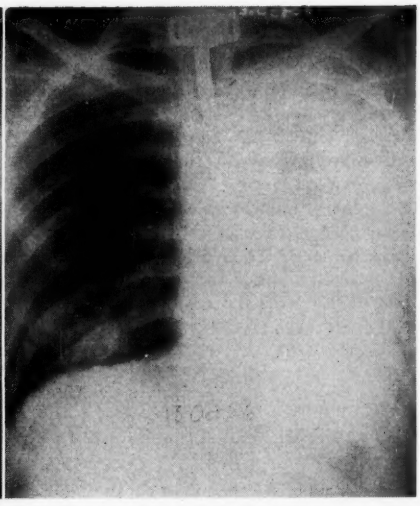


Fig. 3.

Fig. 2..—Oct. 12, 1956. Left intrathoracic tubes were removed after full re-expansion of the left lung and progressive resolution of mediastinal and subcutaneous emphysema. A pneumonic infiltration is conspicuous at the right base.

Fig. 3.—Oct. 13, 1956. There is opacification of the left hemithorax due to a large effusion and atelectasis. In spite of effusion the atelectasis resulted in mediastinal shift to the left. Pneumonia at right base again noted.

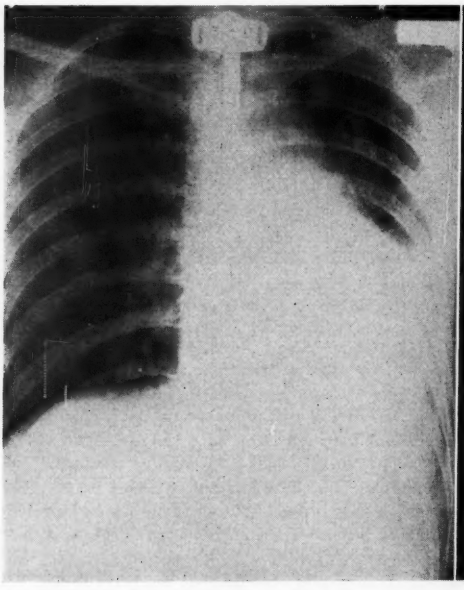
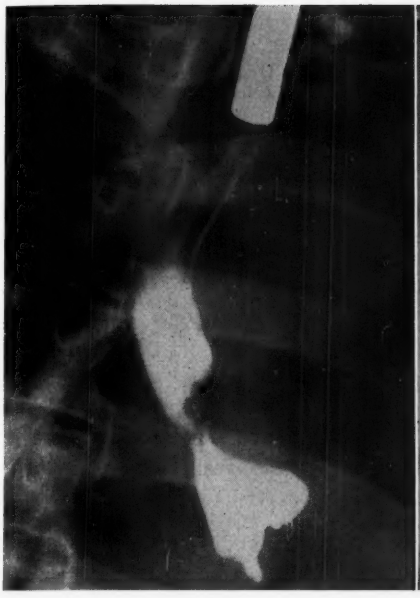


Fig. 4.

Fig. 5.

Fig. 4.—Oct. 17, 1956. The mediastinal shift persists in spite of repeated bronchoscopy and thoracentesis. Right bronchopneumonia cleared.

Fig. 5.—Oct. 17, 1956. Planigram demonstrates obstruction 2 cm. beyond the origin of left main-stem bronchus.



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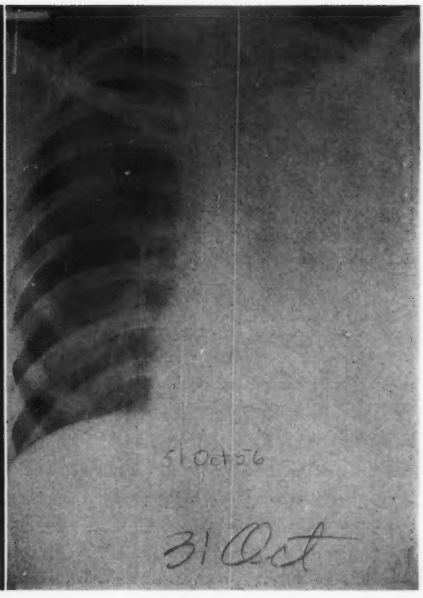

Fig. 7.

Fig. 6.—Oct. 19, 1956. Oily Dionosil, instilled without anesthesia, demonstrates an abrupt impingement on the upper aspect of the left main-stem bronchus for about 1.5 cm. A thin passage along the inferior border is open, but the lobar bronchi beyond could not be filled.

Fig. 7.—Oct. 31, 1956. Recurrent massive left pleural effusions have returned the mediastinal structures to the midline in spite of atelectasis.

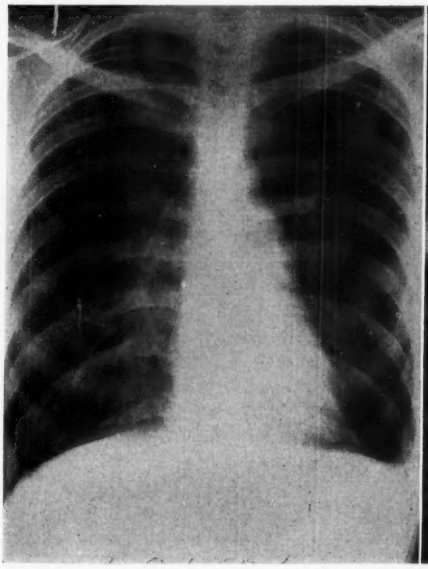

Fig. 8.

Fig. 9.

Fig. 8.—Oct. 8, 1957. The chest is essentially normal except for regenerating left fifth rib and minimal pleural reaction at the costophrenic angle.

Fig. 9.—Oct. 9, 1957. Bronchogram demonstrates the patency of the left main-stem bronchus. The narrowing occurs where two cartilaginous rings were destroyed.

During the next 2 months periodic bronchoscopy was performed to make certain no stricture was forming. The patient's last visit was one year following surgery (Fig. 8) at which time a bronchoscope easily passed the area of anastomosis; however, a bronchogram (Fig. 9) showed indentation on the upper border of the bronchus at the cartilage defect, but without a stricture, as observed at bronchoscopy. Although no preoperative function studies were carried out, one year following surgery the vital capacity was 95 per cent of normal and the maximum breathing capacity was 101 per cent of normal. At the present time he is doing full duty as an aircraft crew chief.

SUMMARY

This case represents a fracture of the main-stem bronchus due to a crushing injury of the chest with a force of injury being transmitted to a closed hollow viscus. It is unusual that the patient experienced no broken ribs or hemoptysis; however, similar cases have been reported.[4] The immediate expansion of the left lung by closed-tube drainage, followed by complete atelectasis the next day can be explained by the entrance of adjacent mediastinal tissue into the fracture site, causing complete stenosis. Planigraphy and bronchography supplemented bronchoscopy to demonstrate the site and length of injury. Repair was performed 20 days following injury. It is suggested that the treatment of fracture of a main-stem bronchus is re-establishment of continuity as soon as clinically possible, so as to prevent bronchiectasis or abscess with the resultant loss of pulmonary functions.

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THE TREATMENT OF APPARENT SOLITARY PULMONARY METASTASES

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IN CARING for patients with malignant neoplasms, evidence of a distant metastasis ordinarily is considered synonymous with uncontrollable disease. In the past few years, there have been several reports of gratifying results following treatment of apparent solitary pulmonary metastases.[1-6] Treatment in these reported cases has been surgical. This is a report of 7 cases in which treatment of apparent solitary pulmonary metastases may have been of value in the care of patients. In one of these, the only treatment of the pulmonary metastatic lesion was by irradiation, while in a second, renewed local progression of the metastatic lesion, following initial response to irradiation, necessitated resection. The other 5 patients were treated initially by resection.

CASE REPORTS

CASE 1.—The patient was a 53-year-old white woman who had had her left forearm amputated 12½ years previously for malignant synovioma involving the wrist. One and one-half years later, a mass was noted in the lower lobe of the right lung (Fig. 1). A right pneumonectomy was performed at that time. The tumor measured 10 by 6 by 5 cm. and was moderately well encapsulated. The pathologic diagnosis was malignant neoplasm consistent with metastasis from the malignant synovioma of the left wrist. This patient remains well 11 years after resection of the pulmonary metastasis.

CASE 2.—This patient was a 60-year-old white woman who had been seen at the Tumor Institute 11½ years previously because of abnormal vaginal bleeding of one month's duration. The cervix was firm and nodular. There was palpable induration and ulceration extending into the left vaginal vault. The pathologic report was adenocarcinoma of the endocervix. Treatment was by means of vaginal and intrauterine radium, followed by 800 kv. roentgen therapy to the pelvis. Nine and one-half years ago, a 1 cm. diameter nodule was noted in the upper lobe of the right lung (Fig. 2). Because this patient was employed in a tuberculosis hospital and there was apparent central calcification in the nodule, observation was recommended. Following x-ray evidence of increase in the size of the nodule (Fig. 3), the upper lobe of the right lung was resected 6 years ago. The pathologic diagnosis of the lung nodule was metastatic adenocarcinoma. This patient is well 6 years after resection of the metastatic lesion and 11½ years after treatment of the primary lesion of the endocervix.

CASE 3.—The patient was a 43-year-old white man who had had an enlarging pigmented lesion of the skin of his left back treated elsewhere by electrocautery 9 years previously. One and one-half years later, a nodule was excised from the left anterior axillary line. This was reported elsewhere as "malignant tissue." This patient was first seen at the Tumor

From the Tumor Institute of The Swedish Hospital.
Received for publication Dec. 18, 1957.

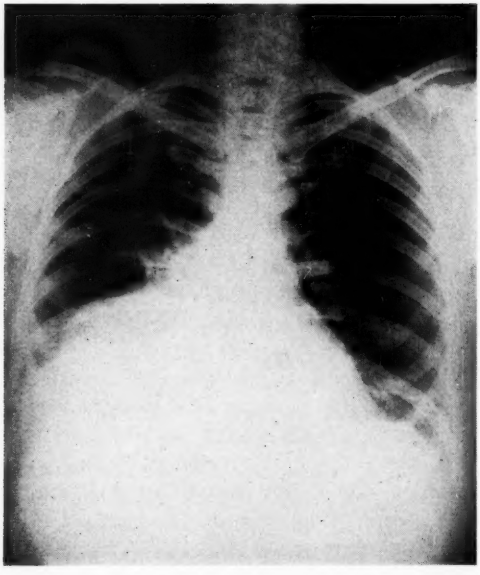
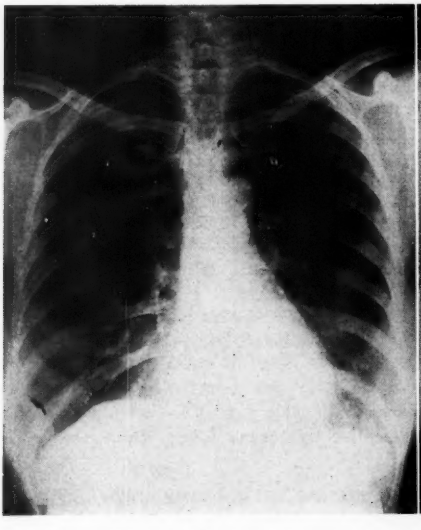


Fig. 1.—Case 1. Metastatic synovioma in the lower lobe of the right lung noted 18 months after resection of malignant synovioma of the left wrist.



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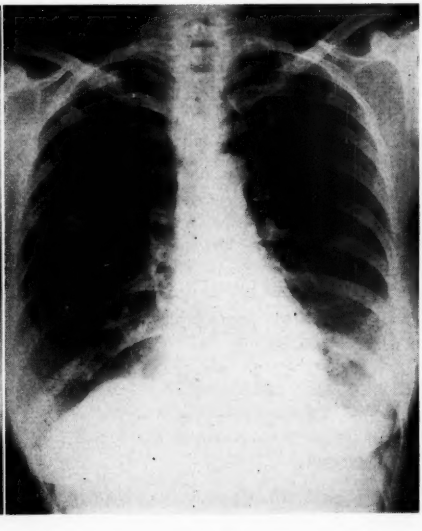

Fig. 3.

Fig. 2.—Case 2. Pulmonary parenchymal nodule noted 9½ years ago, 2 years after treatment of adenocarcinoma of endocervix.

Fig. 3.—Case 2.—Lesion shown in Fig. 2, 3½ years later, when upper lobe of right lung was resected 6 years ago.

Institute 7 years ago. At that time, because of palpable abnormality beneath the scar of the previous excision in the anterior axillary line and in the left axilla, a wide excision, with removal of the pectoralis muscles, and a left axillary dissection were performed. There was metastatic malignant melanoma in the palpable mass and in one of fourteen axillary nodes examined. Two months later, three posterior left chest wall skin nodules were removed. Five years ago, this patient reported to The Swedish Hospital with severe hemoptysis. On chest x-ray, there was evidence of a left hilar mass (Fig. 4). Because of the onset of massive hemorrhage, the exact site of origin of the bleeding could not be determined at bronchoscopy. A left pneumonectomy was performed in an attempt to control this massive hemoptysis. There was a firm mass within the lung, immediately distal to the left hilus.

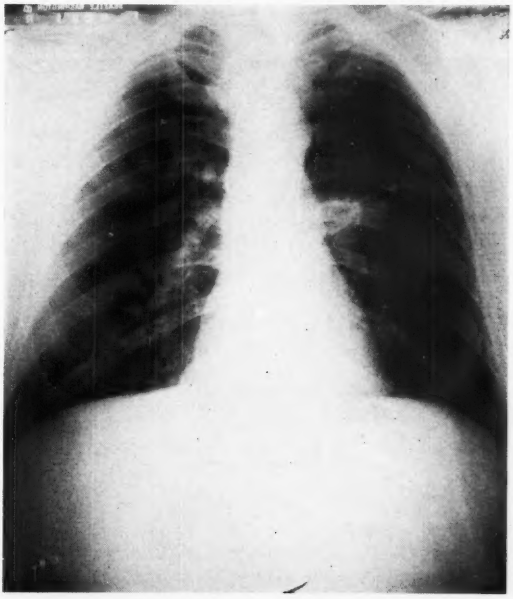

Fig. 4.—Case 3. Metastatic malignant melanoma eroding cartilages of proximal major bronchus of lower lobe of left lung.

On examination, this ovoid tumor, 3 by 2½ by 2½ cm., had eroded bronchial cartilages and was projecting into a major bronchus in the anterior lower lobe. The pathologic diagnosis was metastatic malignant melanoma. There was no histologic evidence of metastatic tumor in thirteen lymph nodes examined. A local recurrence in the scar on his back was excised 1 year ago and, to date, 5 years after pneumonectomy, this patient remains clinically well.

Case 4.—This patient was a 44 year-old-white woman who dated a mass in her right breast from the onset of menses 35 years previously. A second mass in the same breast had been present for the preceding 12 years. The larger mass, first noted in the breast, recently had increased in size prior to her initial examination at The Swedish Hospital. Following a biopsy report of fibrosarcoma from the larger mass, a right simple mastectomy had been performed 6 years previously. The pathologic report of the smaller mass was fibroadenoma. The patient then was followed elsewhere by a physician for whom she worked, until 2 years ago, when she returned with a 6-week history of severe nonproductive cough. At that time, there was x-ray evidence of a mass in the upper lobe of the left lung (Fig. 5). The upper lobe was then resected and it was noted that this 5 by 4 by 2 cm., well-circumscribed tumor extended to the pleural surface. The pathologic diagnosis was metastatic sarcoma.

The patient was clinically well until 6 months later, when she complained of severe left headache. One month later, she was hospitalized elsewhere for this complaint and craniotomy was performed for a tumor in the left cerebral hemisphere which proved to be metastatic. The patient died postoperatively.

CASE 5.—The patient was a 39-year-old white man who had had a left orchiectomy 5 years previously. The pathologic diagnosis was embryonal carcinoma with blood vessel invasion. He was then seen at the Tumor Institute where postoperative 400 kv. roentgen therapy was directed to iliac and periaortic lymphatic pathways to the level of the renal pedicles. The calculated midplane dose was 3,000 r in 47 days. One year later, two nodules, with greatest dimension of 4 cm., were noted in the superior dorsal segment of the lower lobe and lingula of the left lung (Fig. 6). These nodules were treated with 400 kv. x-ray through opposing anterior and posterior fields, 10 by 10 cm. The calculated midplane dose

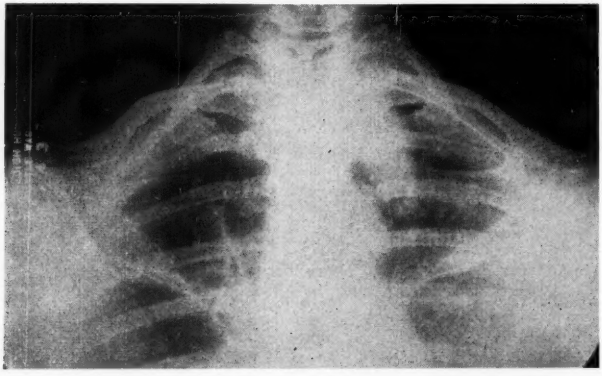

Fig. 5.—Case 4. Metastatic sarcoma in the upper lobe of left lung 4 years after right simple mastectomy.

in the chest was 2,300 r in 20 days. As can be seen on the accompanying x-ray reproduction (Fig. 7), 6 months later the residual lesion in the lung was barely evident. Fourteen months after roentgen therapy, one nodule had regrown to an estimated diameter of 3 cm. (Fig. 8). At that time, the lower lobe of the left lung was resected. The pathologic diagnosis was metastatic embryonal carcinoma. This patient remains well 30 months after pulmonary resection.

CASE 6.—The patient was a 26-year-old white man who had had a right orchiectomy 3½ years previously. The pathologic diagnosis was malignant teratoma with embryonal carcinoma. He was seen at the Tumor Institute postoperatively, where 400 kv. roentgen therapy was directed to the right iliac and periaortic lymphatic pathways to the level of the renal pedicles. The calculated midplane dosage was 2,500 r in 43 days. Six months later, there were at least two nodules, with greatest dimension of 3 cm., in the right midlung as visualized on x-ray studies (Fig. 9). The involved portion of the lung was treated with 2 MEV roentgen therapy through opposing anterior and posterior circular fields, 8 cm. in diameter, with a calculated mid-plane dose of 3,000 r in 29 days. The lesion visibly disappeared on x-ray within 6 months. Three years later, the lungs appear normal in x-ray studies of the chest (Fig. 10). It should be noted that in this case, as in the other in which roentgen therapy was directed to the lung, there is no x-ray evidence of pulmonary parenchymal damage.

CASE 7.—This patient was a 57-year-old white man who was first seen in the Tumor Institute 2½ years ago. He had had a right orchiectomy 9 years before. The pathologic diagnosis at that time was interstitial cell hyperplasia of the testis with a question of

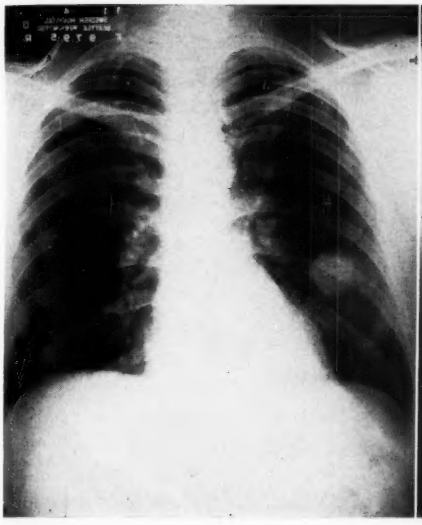

Fig. 6.

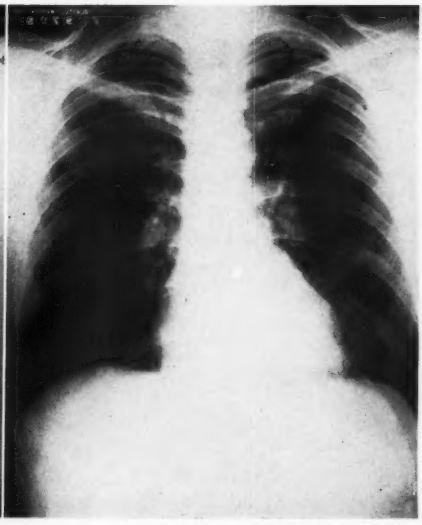

Fig. 7.

Fig. 6.—Case 5. Metastatic embryonal carcinoma in lingula and lower lobe of left lung 1 year after orchiectomy and postoperative irradiation.

Fig. 7.—Case 5. Minimal residual abnormality 6 months after roentgen therapy to pulmonary metastases.

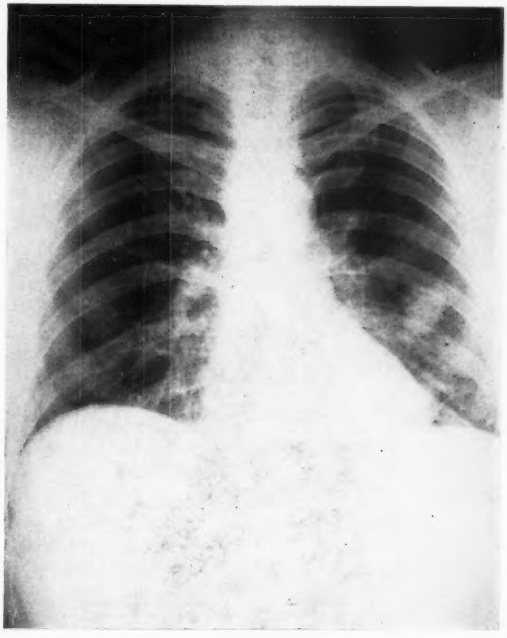

Fig. 8.—Case 5. Regrowth of pulmonary metastatic embryonal carcinoma 14 months after local roentgen therapy.

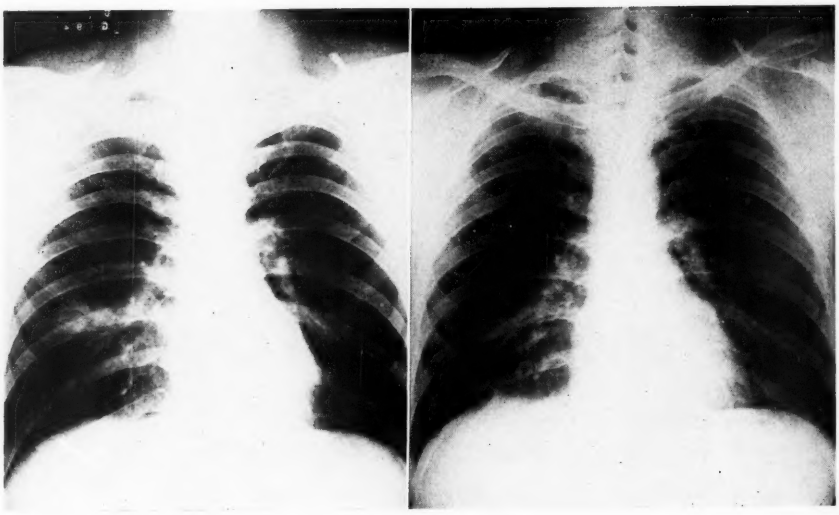

Fig. 9. Fig. 10.

Fig. 9.—Case 6. Metastatic embryonal carcinoma in teratoma 6 months after orchiectomy and postoperative irradiation.

Fig. 10.—Case 6. Normal appearance of chest 3 years after roentgen therapy for metastatic embryonal carcinoma.

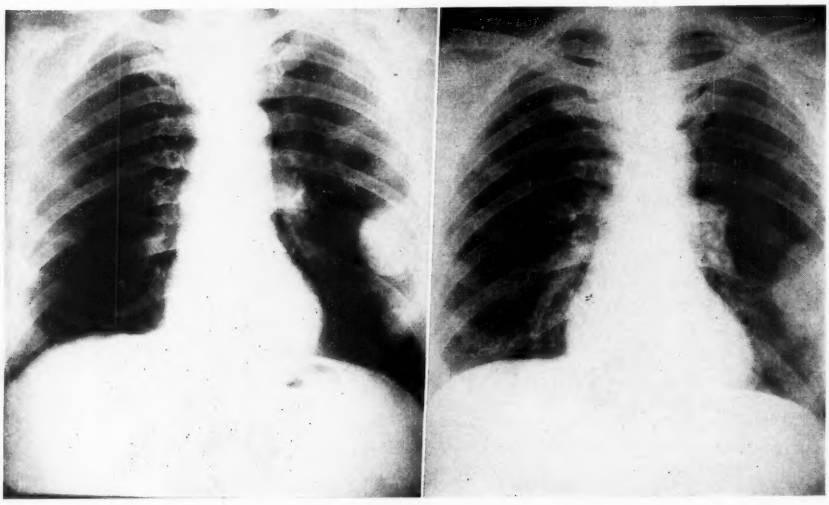

Fig. 11. Fig. 12.

Fig. 11.—Case 7. Metastatic interstitial cell carcinoma of the testis, 6½ years after orchiectomy.

Fig. 12.—Case 7. Metastatic nodules with evidence of enlargement during the 6-month interval since Fig. 11 was made.

teratoma. One year prior to initial admission at the Tumor Institute, he had noted bilateral gynecomastia which had progressed until the enlarged breasts were of definite female appearance. Six months prior to admission, three circumscribed pulmonary metastases in the left lower lung were discovered (Fig. 11). These had increased slightly in size in this interval (Fig. 12). At time of admission, the Aschheim-Zondek test was negative. Twenty-four-hour urine 17 ketosteroids were 12.2 grams. The test for FSH was negative at about six mouse units with no definite end point reached. The tracheobronchial tree was normal at bronchoscopy. Two and one-half years ago, lingulectomy and wedge resection of the posterior basal segment of the lower lobe of the left lung were performed. The well-circumscribed tumors measured 8 by 6 by 3 cm. and about 2 cm. in diameter in the lingula, and 6 by 3 by 2 cm. in the posterior basilar segment. The pathologic diagnosis was metastatic interstitial cell carcinoma in the lung. Presently, this patient is without evidence of disease. The gynecomastia has partially resolved in the interim.

DISCUSSION

Presentation of these cases is not an endorsement of ill-considered onslaughts on pulmonary metastases. Usually, the appearance of a pulmonary metastasis, regardless of possible control of the primary lesion and the interval preceding the clinical appearance of the metastasis, is considered synonymous with incurability. In each of these 7 cases, representing varying forms of cancer in patients of varying ages, treatment of the pulmonary metastases has been of value in the management of the patient, whether or not permanent clinical freedom from disease has been accomplished. The 2 young men with testicular neoplasms (Cases 5 and 6), both with young families, have had 2½ to 3 years of good health since the treatment of the metastases. Two women (Cases 1 and 2) remain well 6 and 11 years, respectively, after treatment of this usually fatal development. One man (Case 3) has remained functional for 5 years after pulmonary resection for intractable hemoptysis, although he is not considered free of disease. In Case 4, the accomplishment is open to question, although the patient was asymptomatic for 6 months until sudden terminal illness with cerebral metastasis.

These cases illustrate the vagaries constantly present in the evaluation and treatment of patients with malignant tumors and they again emphasize the necessity for treating each patient upon an individual basis.

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POSTOPERATIVE EAR OXIMETER STUDIES ON PATIENTS WHO HAVE UNDERGONE PULMONARY RESECTION

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THIS STUDY was undertaken to determine the effect of pulmonary resection on the blood oxygen saturation of patients during the postoperative period. We also wanted to see if this measure of oxygenation could be correlated with clinical findings, and to determine whether ear oximeter readings would indicate the status of pulmonary function following thoracotomy. It was also thought desirable to see if the use of the ear oximeter could be of value in predicting impending complications, before more cumbersome diagnostic aids were used.

Maier and Cournand[1] reported, in 1943, a series of 38 patients, on whom arterial blood oxygen saturation was done after pulmonary resection, by the Van Slyke and Neill[2] manometric method. In 1954, Björk and Hilty[3] reported a series of 32 patients, on whom the arterial blood oxygen tensions were determined by the method of Riley, Proemmel, and Franke[4] after pulmonary resection.

These reported studies are rather difficult technically, and the blood samples were taken at irregular intervals during the postoperative period. The direct-reading ear oximeter,† which was used in our study, is relatively simple to operate, requiring only about 5 to 7 minutes per patient to obtain the blood oxygen saturation. The method is painless and of little bother to the patients. If one appeared daily before a postoperative patient to do an arterial puncture, considerable objection to that procedure probably would develop before 10 days had passed. Our study appeared more practical, and we were able to do tests for all 62 patients daily, and a few several times a day, with no resistance or objection at any time. In fact, a good number of the patients appreciated the extra care when the purpose of the test was explained to them!

Earl Wood,[5] in 1949, described the ear oximeter, which records absolute values accurate to within ±3 per cent. Stephen and co-workers[6] reported the

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Received for publication Jan. 31, 1958.
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average discrepancy between blood oxygen saturation as measured by the Van Slyke manometric method and the oximeter values at about ±2.5 per cent in 100 patients with normal circulation.

METHOD

The blood oxygen saturation was determined by ear oximetry daily for 10 days postoperatively in a series of 62 patients who had various types of resections for tuberculosis. In this series, most of the preoperative readings were taken just prior to induction of anesthesia, and after the patient had received only a belladonna derivative, usually scopolamine, for premedication. A few readings were taken the day prior to operation. The oximeter was used as a monitor during the operation, but these results are not included in this study. A reading was taken on each patient the afternoon of the operative day when the patient was awake and responding. Determinations were made at least once daily, usually between 3:00 P.M. and 4:00 P.M. or between 10:00 A.M. and 11:00 A.M. for 10 days.

Operations performed on these patients included 29 segmental resections, 20 lobectomies, 3 pneumonectomies, 2 decortications, 4 space-filling procedures, 1 closure of a bronchopleural fistula, and 3 open drainages. Two patients in this series died within the 10-day period. Five patients of the group studied had postoperative atelectasis, severe enough to require treatment.

The average blood oxygen saturation for normal subjects breathing room air is 97 per cent, ranging from 92 to 102 per cent. Our oximeter was calibrated on 12 normal subjects, breathing room air, and found to average 96.5 per cent.

RESULTS

The pre-induction readings in this series of patients ranged from 85 per cent to 100.5 per cent with an average of 93 per cent. The lower average in this series is attributed to lowered pulmonary function due to disease, rather than premedication. As a control, 38 patients with varying stages of pulmonary tuberculosis, who had not had thoracic operations, were checked by ear oximetry. The average in this control series was also 93 per cent, ranging from 88 to 97 per cent.

Oxygen, by nasal catheter, is routinely administered to our patients for a period of 12 to 24 hours postoperatively, and longer in a few patients requiring it. Accordingly, the readings taken on the operative day are generally higher, the average being 97 per cent. The lowest readings occurred on the second and third postoperative days, when the average was 89.5 per cent. The blood oxygen saturations then gradually returned to near normal levels, for this group of patients, and were at preoperative levels by the seventh to the tenth postoperative day.

DISCUSSION

In some cases these observations correlated well with other clinical aids, such as x-ray and blood gas studies. The 5 patients who developed atelectasis,

severe enough to require bronchoscopy or tracheal aspiration, had an average oximeter reading of 87 per cent on the second, third, and fourth days, and an average of 90 per cent on the fifth day, at which time the atelectasis usually had cleared. In other patients, the low readings could be accounted for by the fact that the patient had poor pulmonary function before and after operation. Some patients had severe pain during breathing, coughing, or moving and a very few low readings could be attributed to depressed respiration from the effect of opiates.

There were a few low oximeter readings for which the cause could not be determined. At the present time we are using the method of Alexander and Reydman,[7] described in 1953, for measuring the oxygen resaturation time in order to determine whether oxygen diffusion may be a factor in these unexplained low readings. We are also measuring the total and the timed vital capacities on the same patients during the postoperative period.

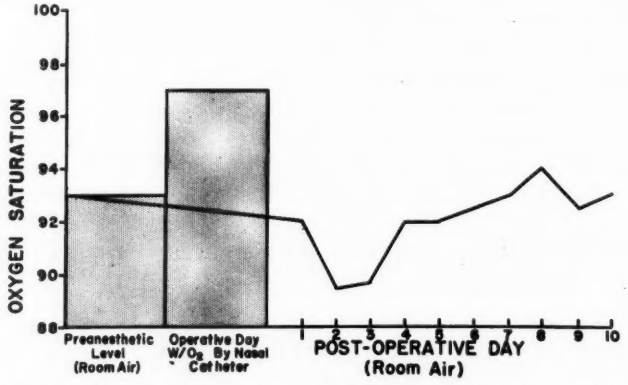

Fig. 1.—Average oxygen saturation (by ear oximeter) in patients after thoracic operations.

In most cases there seemed to be very little correlation between oximeter readings and pulse rates. Pulse rates varied with elevation of temperature, excitement, pain, and exertion. Hypoxia had to be quite severe before elevations in pulse rate occurred in these patients, most of whom were lying quietly in bed the first few postoperative days, except during the times they were encouraged to sit up, cough, and take deep breaths.

In conclusion, it was found that the blood oxygen saturation of this series of 62 patients was lowest on the second and third postoperative days with a gradual return to basal levels by the seventh or eighth day (Fig. 1). In patients who had obvious respiratory complications, the readings were below average.

It is thought the ear oximeter can be useful as a guide in evaluating pulmonary function in the postoperative period following thoracotomy. We found occasional instances in which severe pulmonary complications could be predicted from the oximetry readings. Oximetry does not indicate a specific

diagnosis, and there were instances in which patients exhibited low oxygen saturation when we were unable to discover any pulmonary or circulatory complications.

Further studies, including other pulmonary function tests, are in progress in an effort to determine the cause of these few unexplained low blood oxygen saturations.

SUMMARY

Sixty-two patients undergoing intrathoracic operations were studied in regard to arterial blood oxygen saturation. These were made by means of an ear oximeter for 10 days following operation. The second and third postoperative days produced the lowest readings, with return to normal values by the seventh to the tenth day.

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THE SURGICAL TREATMENT OF CHRONIC PLEURAL EMPYEMA

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IN RECENT years the prognosis of chronic pleural empyema has improved to a remarkable extent. Although much of this improvement is attributable to the use of potent antibacterial agents, other factors undoubtedly have contributed to these results. In attempting to evaluate some of these factors, one discovers that it is difficult to gain an adequate perspective from recent reports,[1-12] as they are generally limited in scope and occasionally controversial. As it seemed likely that the experience in a hospital for thoracic disease might provide such information, it was decided to review the cases of chronic empyema treated surgically at the Ray Brook State Tuberculosis Hospital between Jan. 1, 1950, and Oct. 1, 1957.

To provide a basis for comparison, both tuberculous and pyogenic empyemas were included in this study. Several sterile empyemas were also included. Patients treated merely by thoracentesis or surgical drainage were excluded. Thus, there were available for study 45 patients treated by 56 definitive surgical procedures. Thirty-nine patients had pulmonary or pleural tuberculosis at one time or another. The remaining 6 patients had, in addition to empyema, the following diseases: 1, cystic disease of the lung; 1, bronchogenic carcinoma; 1, spontaneous pneumothorax; 2, pyogenic pneumonia; and 1, pulmonary disease of undetermined etiology. The empyemas followed thoracotomy in 29 instances; 9 complicated therapeutic or spontaneous pneumothorax; and 10 were primary. When first bacteriologic studies were performed at Ray Brook, 23 empyemas yielded tubercle bacilli, 8 yielded mixed tuberculous and pyogenic organisms, 11 yielded pyogenic organisms, and 3 yielded purulent material sterile on culture. The pyogenic infections usually included staphylococci. By the time the surgery listed in Table III was undertaken, the number of tuberculous and mixed tuberculous and pyogenic infections had decreased to 16 and 7, respectively; and the number of pyogenic infections and sterile pleural spaces had increased to 13 and 9, respectively.

Bronchopleural Fistula.—Bronchopleural fistula was present at one time or another in 34 of the 45 patients. Bronchopleural fistula seemed to have no effect upon the results of treatment, often healing spontaneously with adequate drainage or drug therapy. Except in pulmonary decortication where leakage of air from the lung could interfere with postoperative pulmonary expansion,

From the Ray Brook State Tuberculosis Hospital, Ray Brook, N. Y.
Received for publication Dec. 21, 1957.

it seemed to matter little whether or not bronchopleural fistulas were sutured, provided the pleural space was obliterated and adequate amounts of tissue were available to cover the fistula. Pulmonary resection merely to eradicate a bronchopleural fistula is not considered necessary.

Types of Infection.—It is interesting to note that of the 7 patients with mixed tuberculous and pyogenic empyema at the first bacteriologic examination, 6 had already been subjected to thoracotomy and 1 had received a recent bronchogram, a possible source of contamination of the pleural cavity. Of the 14 patients with pure pyogenic empyemas at the first bacteriologic examination, 11 had been submitted to previous chest surgery. These findings are especially interesting since we have observed pleural spaces communicating with the bronchial tree for prolonged periods without detecting evidence of infection.[13] It would appear, therefore, that secondary pyogenic infection is frequently iatrogenic, a belief which is certainly not new.

From Table I it may be inferred that tuberculous empyemas frequently can be rendered sterile by drug therapy alone. Almost all will respond favorably to drug therapy combined with adequate surgery. On the other hand, surgery without adequate antituberculous drug therapy is not often successful.

TABLE I. DRUG REGIMENS EMPLOYED WITH SURGERY IN THE TREATMENT OF TUBERCULOUS AND MIXED EMPYEMAS

EFFECTIVE DRUG REGIMENS	NUMBER OF EMPYEMAS TREATED	EMPYEMAS YIELDING CULTURES NEGATIVE FOR TUBERCLE BACILLI	
		BEFORE DEFINITIVE SURGERY	AFTER DEFINITIVE SURGERY
PAS	1	0	1
INH	4	1	1
SM, PAS	4*	2	2
SM, INH	2	1	1
PAS, INH	4	2	2
SM, PAS, INH	2	1	1
INH, cycloserine	1	1	0
PZA	6	0	4
VIO	1	0	0
VIO, PZA	4†	0	3
VIO, cycloserine	1	0	1
No drugs	4	0	1
Total	34	8	17

PAS—para-aminosalicylic acid; INH—isoniazid; SM—streptomycin; PZA—pyrazinamide; VIO—viomycin.

*One patient received these drugs only by instillation into the pleural cavity.

†One patient had previously received 4 months of pyrazinamide.

The prognosis for mixed tuberculous and pyogenic empyema is no longer as poor as it was prior to the introduction of antituberculous drug therapy.[14] The presence of secondary pyogenic infection does not seem to affect the response of the tuberculous infection to antituberculous drug therapy. In Table II, the apparent superiority of the results of treatment of tuberculous compared with mixed infections can be explained upon the basis of drug regimens and type of surgery employed.

TABLE II. RESULTS OF TREATMENT

BACTERIOLOGY OF EMPYEMA PRIOR TO TREATMENT	NUMBER OF PATIENTS	CONDITION AT LAST EXAMINATION				
			RESULTS OF CULTURES			
		CURED	MIXED EMPYEMA	TUBERCULOUS EMPYEMA	PYOGENIC EMPYEMA	DEAD
Tuberculous	23	19		1	1	2*
Pyogenic	11	11				
Mixed	8	4	1		1	2*
Negative culture	3	3				
Total	45	37				4

*Only one patient died of causes directly related to tuberculosis.

Chronic pyogenic empyemas in our experience have rarely been rendered permanently sterile by drugs as long as a pleural space existed. This may have been due to the early appearance of drug-resistant organisms. On the other hand, these empyemas have been cured easily by surgery with little or no antibacterial therapy, provided the surgery obliterated the pleural space.

Antituberculous Drug Therapy.—Antituberculous drug therapy is reviewed in Table I. Effective drugs are defined as drugs to which tubercle bacilli are susceptible. Organisms are considered susceptible if they do not grow in the presence of drug in vitro or if the patient has not received the drug previously. Concentrations of drug employed are 10 mcg. of streptomycin, 10 mcg. of para-aminosalicylic acid, and 5 mcg. of isoniazid. Sensitivities to pyrazinamide, viomycin, and cycloserine are not performed routinely. Although there are too few cases to provide statistical information regarding the various regimens, it is evident that it was possible to render the empyemas noninfectious with drugs alone in many instances. One is impressed by the fact that, in these instances, isoniazid was almost always one of the drugs employed. This seems to support some of the recently published opinions regarding the superiority of isoniazid in combination with other drugs.[1, 15]

Complications and Mortality.—Serious postoperative complications consisted of massive intrapleural hemorrhage and contralateral spontaneous pneumothorax, both in the same patient. This patient later died of progressive pulmonary tuberculosis and mixed tuberculous and pyogenic empyema. One additional patient died of similar causes. It is believed that these fatalities are attributable to the use of a poor drug regimen in one instance (viomycin), and early cessation of effective drug therapy in another instance (patient left the hospital 2 weeks postoperatively). One patient died of cor pulmonale before her mixed empyema could be cured. Another patient with tuberculous empyema died following abdominal surgery for ulcerative colitis. The immediate cause of death was hemorrhage from a gastric ulcer.

Surgical Considerations.—Surgery was performed in 56 instances. The various types of surgery are listed in Table III. Standard thoracoplasty consisted of subperiosteal removal of the ribs over the empyema space. Polyethylene beads were inserted subcostally in the plombage thoracoplasties. The modified Schede procedures were generally similar to the type described by

TABLE III. RESULTS OF DEFINITIVE SURGERY

PROCEDURE	NUMBER OF OPERATIONS	RESULTS OF CULTURES OF EMPYEMA AT OPERATION				CONDITION AT LAST EXAMINATION*					DEAD
		STERILE SPACE	MIXED EMPYEMA	TUBERCULOUS EMPYEMA	PYOGENIC EMPYEMA	CURED	STERILE SPACE	MIXED EMPYEMA	TUBERCULOUS EMPYEMA	PYOGENIC EMPYEMA	
Standard thoracoplasty	9	2	1	6		5	1		2		1
Plombage thoracoplasty	2			2		1			1		
Modified Schede	15	1	2		12	13		1		1	
Decortication	9	3	1	4	1	8					1
Decortication and thoracoplasty	4	2			2	2	1			1	
Decortication and resection	8	2	1	4	1	5			1	1	1
Decortication, resection, and thoracoplasty	3	1		2		3					
Plastic procedure	6		2	1	3	4				1	1
Total	56	11	7	19	19	41	2	1	4	4	4

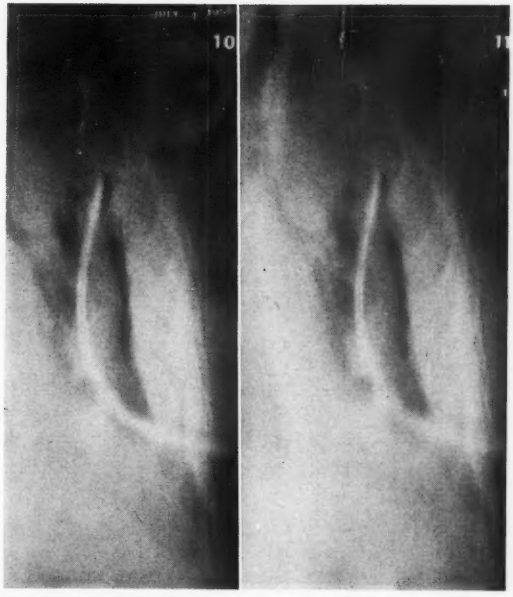
*Some of the failures were later cured by other procedures.

Grow.[16] Pulmonary decortication was performed both with and without removal of the parietal pleura. Occasionally, pulmonary resection was employed in combination with some other procedure. Two successive procedures were performed on 8 patients, and 3, on 3 patients.

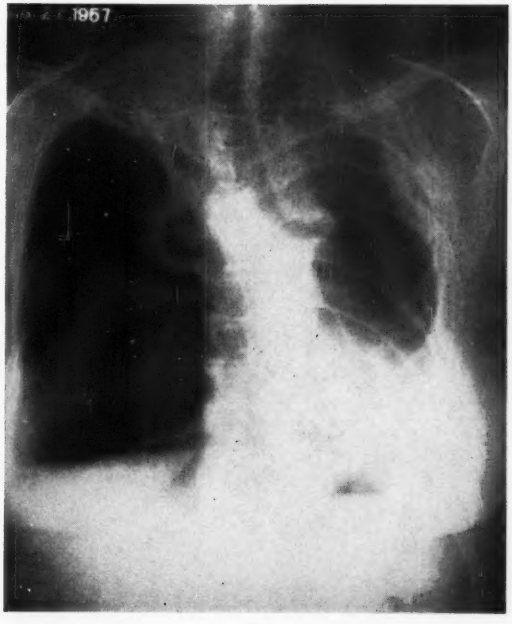
To be successful, definitive surgery should obliterate the pleural space. A persistent pleural space, even if sterile, is a potential invitation to infection. Standard and plombage thoracoplasty are of limited value, and are useful only when the walls of the empyema are not very rigid. Plombage thoracoplasty is occasionally complicated by infection in the subcostal space. On the other hand, the Grow modification of the Schede procedure can be used with little fear of deformity and with excellent results; being confined to the diseased area, it is well tolerated by the poor-risk patient. It can be performed in stages, or repeated, should the first procedure fail to achieve the expected result. It is important to remove foreign bodies such as surgical silk or calcium deposits, as these may be a source of chronicity. A simple method of removing calcium is curettage. The wound may be sutured almost completely at the time of surgery. Adequate drainage should be maintained with a soft rubber tube until the empyema space has obliterated itself from the depths outward. A pressure dressing for several weeks will foster early obliteration of the space.

Decortication has proved most useful in expanding lungs which contain little disease and where the empyemas have been of relatively short duration or small size. Appreciable pulmonary fibrosis is a well-known cause of poor postoperative re-expansion. Long-standing empyema may involve the underlying lung so as to produce a similar effect and, if the empyema involves a large proportion of the lung surface, postoperative re-expansion of the lung may be poor. On the other hand, if the empyema involves only a small portion of the lung surface, the remaining lung will often expand appreciably if it is completely freed, including the fissures. A poorly expansile lung may be useful as a space filler (Fig. 1). For this reason resection of such a lung is rarely performed with decortication, except for indications applicable in the absence of empyema. Although parietal decortication would seem generally desirable, it does not appear necessary to effect a cure of empyema. Parietal pleura may be allowed to remain if one wishes to minimize blood loss. Occasionally in these instances pleural symphysis may be delayed, but this does not seem serious. Pulmonary expansion following decortication appears to have no ill effects upon pulmonary tuberculosis, provided adequate antituberculous therapy is administered.

It has been found useful to employ a number of technical aids when performing decortication. As in other types of pulmonary surgery, the Bovie coagulation unit is an excellent hemostatic agent. A 2-inch dental type mirror often exposes to vision the most remote recesses of the pleural cavity. A curved pair of scissors (Fig. 3) can be used to cut *"around corners,"* with or without the aid of the mirror. In a number of instances, 2 or even 3 separate intercostal incisions at different levels have provided excellent exposure to otherwise inaccessible portions of the pleural cavity.



A.



B.

Fig. 1.—Successful decortication for mixed tuberculosis and pyogenic empyema illustrating the use of a poorly expansile lung as a space-filler. About one year postoperatively, the vital capacity had increased by 8 per cent of the preoperative value. *A*, Preoperative tomogram. *B*, Postoperative roentgenogram.

A.

B.

Fig. 2.—Successful obliteration of a mixed tuberculous and pyogenic empyema by a modified Schede thoracoplasty. *A*, Preoperative bronchogram indicating bronchopleural fistula. *B*, Preoperative lateral tomogram outlining the empyema space.

TABLE IV. EFFECT OF DECORTICATION UPON VITAL CAPACITY

PATIENT	EXTENT OF DECORTICATION	VITAL CAPACITY			
		PRE-OPERATIVE (C.C.)	POST-OPERATIVE (C.C.)	POST-OPERATIVE INTERVAL IN DAYS	PER CENT POST-OPERATIVE CHANGE
L. B.	Visceral + parietal	2,000	1,000 to 2,550	6 to 366	−50 to +28
C. M.	Visceral + parietal	2,200	2,900	23	+20
I. B.	Visceral + parietal	1,200	1,100 to 1,300	30 to 690	−8 to +8
G. G.	Visceral + parietal	2,200	2,200 to 2,200	22 to 444	0
M. M.	Visceral	1,500	1,450 to 1,800	29 to 151	−3 to +20
L. G.	Visceral	1,900	2,000 to 2,800	41 to 447	+5 to +48
L. S.	Visceral	2,300	2,600 to 3,000	15 to 56	+13 to +30
F. B.	Visceral + lobectomy	1,400	1,100 to 1,400	74 to 362	−21 to 0
C. W.	Visceral + lobectomy	3,100	2,400 to 2,600	27 to 43	−33 to −16

Function.—The effect of pulmonary decortication upon vital capacity is shown in Table IV. From this data, functional superiority cannot be claimed for visceral and parietal decortication. As indicated by Patton and associates,[9] the amount of functional gain after decortication is related to the relative increase in expansion of the lung operated upon. The early postoperative vital capacity represents the difference between the depression of ventilation caused by the "thoracotomy effect"[17, 18] and the improvement in ventilation due to pulmonary re-expansion. In this group of patients, decortication alone was accompanied by a permanent increase in vital capacity in all but one instance. Furthermore, improvement in vital capacity occurred in 2 patients despite duration of pulmonary collapse of 22 years and 14 years, respectively.

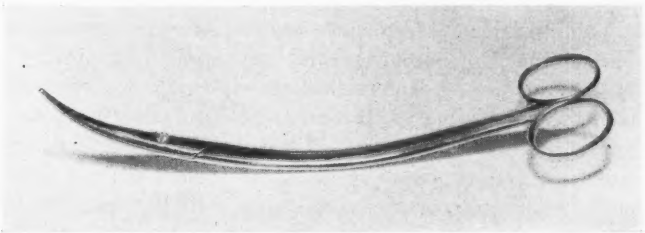


Fig. 3.

Table V represents the effect on vital capacity of the various types of thoracoplasty employed to obliterate pleural spaces. It has been shown that thoracotomy produces a much more profound early depression of vital capacity.[18] Indeed, thoracoplasty performed to obliterate a rigid pleural space seems to have little effect upon vital capacity. There appears to be no correlation between changes in vital capacity and the number of ribs involved in the thoracoplasty procedures. This is to be expected since these procedures usually did not alter the rigidity of the visceral pleura, and they rarely collapsed functioning pulmonary tissue.

There appears to be little evidence to indicate that distention of pulmonary tissue of a degree encountered following decortication can cause emphysema.

Timed vital capacity following decortication[18] shows no early change. It has been interesting to discover well-developed bullous emphysema in several lungs which we have decorticated after they had been collapsed for many years. This would seem to indicate that, at least in these instances, intrapulmonary factors were more important than distention and that collapse did not prevent these factors from causing emphysema.

TABLE V. EFFECT OF THORACOPLASTY UPON VITAL CAPACITY

PATIENT	PROCEDURE (NUMBER OF RIBS)	VITAL CAPACITY			
		PRE-OPERATIVE (C.C.)	POST-OPERATIVE (C.C.)	POST-OPERATIVE INTERVAL IN DAYS	PER CENT POST-OPERATIVE CHANGE
J. W.	Plombage (6)	2,700	2,110 to 2,200	1 to 54	−22 to −18
W. K.	Plombage (4)	1,600	1,400 to 1,600	15 to 240	−12 to 0
M. M.	Plombage (5)	1,400	1,300 to 1,400	3 to 143	−7 to 0
C. G.	Mod. Schede (9)	2,100	2 000	30	−5
W. K.	Mod. Schede (2)	1,550	1,500	36	−3
O. B.	Mod. Schede (2)	3,100	3,200 to 3,000	48 to 198	+3 to −3
E. C.	Standard (5)	1,650	1,550 to 1,700	4 to 28	−6 to +3
J. C.	Standard (5)	1,750	1.600 to 1,650	9 to 66	−8 to −6
F. T.	Standard (9)	1,100	1,000 to 1,400	29 to 215	−9 to +27
A. S.	Standard (2)	2,800	2,800 to 2,800	26 to 45	0
D. O.	Standard (3)	2,100	1,900 to 1,800	17 to 57	−9 to −14
C. M.	Standard (6)	2,400	2,000 to 2,100	7 to 31	−17 to −12
E. K.	Standard (10)	950	900 to 1,000	4 to 52	−5 to +5
I. R.	Standard (6)	950	1,000 to 1 000	15 to 304	+5

DISCUSSION

Recent publications appear to have overemphasized the value of decortication in the treatment of chronic pleural empyema. Undoubtedly there are many advantages in the use of decortication, especially improvement of function and avoidance of deformity. However, there are many instances in which a carefully performed thoracoplasty will accomplish the same functional and curative results as decortication. Perhaps the most important difference between decortication and thoracoplasty lies in the early postoperative ventilatory changes, which may be severely depressed after the former and is usually only slightly altered after the latter. Considerations such as these may influence a surgeon to prefer thoracoplasty to decortication in poor-risk patients.

Drug therapy is undoubtedly responsible for the excellent results of surgery for chronic tuberculous empyema. Surgery is relatively ineffective without such therapy. On the other hand, drug therapy may render tuberculous empyemas noninfectious; but, in many instances, surgery must be combined with drug therapy to effect a cure.

The treatment of chronic pyogenic empyema seems to be dependent more upon surgery than antibacterial agents. With little or no drug therapy, surgery can effect a cure in almost every instance. On the other hand, drugs in the presence of a persistent pleural space are rarely curative probably because of the rapid appearance of drug-resistant organisms.

SUMMARY

A review of almost 7 years' experience with chronic empyema reveals that with proper treatment the prognosis is excellent regardless of the type of infection. Pulmonary decortication and the various types of thoracoplasty are all useful procedures. The indications for these procedures are briefly discussed.

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STRANGULATED DIAPHRAGMATIC HERNIA

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DIAPHRAGMATIC hernias are often considered relatively benign lesions which can be watched with impunity. However, complications do occur in a number of these patients and these can be very serious or even fatal. We, therefore, suggest that a more aggressive attitude be taken in treating this type of hernia. We are reporting here a survey, covering the 10-year period from Jan. 1, 1947, to Jan. 1, 1957, of all cases of strangulated diaphragmatic hernias at the John Gaston Hospital and the Baptist Memorial Hospital, Memphis, Tenn. One or 2 cases were excluded in which the diagnosis was not definitely established. Each hospital had approximately a 500 bed census during the 10-year period.

For the purpose of our paper we are considering a strangulated diaphragmatic hernia as one in which the hernial ring is constricting the herniated organ so as to compromise or cut off the blood supply, either venous or arterial, of the herniated organ or organs. By incarceration of a diaphragmatic hernia we mean that the herniated organs are so bound by adhesions that the hernia is irreducible by medical methods and that surgery is necessary for the reduction of such a hernia.

CASE REPORTS

CASE 1.*—J. W., a white woman aged 74, was admitted Oct. 19, 1948, to the Baptist Memorial Hospital with a diagnosis of incarcerated diaphragmatic hernia. From her history it was evident that she had been known to have a hernia for 20 years as proved by numerous serial films. It was believed that the stomach and colon were in the left chest. For 20 years she had "eaten like a bird."

She had had symptoms of obstruction on numerous occasions, which would usually subside in a few hours after vomiting. Two days before her admission here she began to have left epigastric pain with some vomiting and she noticed she passed relatively little gas by rectum. She also noted the pain sometimes radiated to the left shoulder. On admission she was afebrile and her blood pressure was 120/80 mm. Hg. She was an elderly white woman appearing acutely ill, vomiting intermittently. The heart was normal. The right chest was clear. On the left, breath sounds were absent. The abdomen revealed some tenderness in the left upper quadrant, but no rigidity. A small amount of barium was given by mouth and it was demonstrated that only a small portion of the cardiac end of

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Received for publication Jan. 6, 1958.

*This case was abstracted briefly by Dr. Duane Carr.[2]

the stomach was in the abdomen. Some of the barium could be passed into the supradiaphragmatic portion of the stomach by laying the patient in the prone position with the head down. None of the barium could be encouraged to proceed into the duodenum. The x-ray films also showed a portion of the colon in the left hemithorax.

Despite the fact that the patient's age and general condition created a greater than average risk, a left diaphragmatic herniorrhaphy was performed on Oct. 20, 1948, after proper preparation.

At operation, the stomach, colon, and a portion of the omentum were found herniated into the left hemithorax through a diaphragmatic opening which was approximately 8 cm. in diameter. The edge of the ring was markedly thickened and fibrotic. The posterior part of the diaphragm was very much attenuated and contained no muscle fibers. The thoracic portion of the stomach was hugely dilated and the loop of transverse colon which was also present in the thorax was adherent to the surface of the stomach. In the course of the operation a moderate amount of clear yellow peritoneal fluid was aspirated. The diaphragmatic hernial orifice was lateral to the esophageal hiatus and gave every appearance of being a congenital hernia. The stomach and colon were slightly dusky at first but the colon soon improved when the hernial ring was opened. The hernial sac was closed with interrupted sutures of No. 2-0 cotton. The weakened area posteriorly in the diaphragm was reinforced by imbricating the diaphragm upon itself. The excess portion of the hernial sac was excised. The thoracic wound was closed in layers using interrupted cotton sutures throughout. The patient received 2,500 c.c. of whole blood during and immediately after the operation and was returned to her room in good condition.

The patient tolerated the operation exceedingly well, her pulse rate at no time rising above 90. The blood pressure remained stable at about 126/66 mm. Hg. On Oct. 23, 1948, a left thoracentesis yielded 450 c.c. of thin bloody fluid. The patient had some mild abdominal distention and was slightly dusky at times and irrational. We later discovered that part of her disorientation was apparently due to small doses of Nembutal which were also causing insomnia. The symptoms subsided when the barbiturates were discontinued. She was discharged from the hospital on Nov. 19, 1948, one month after admission.

For the first time in over 20 years she could eat comfortably without regurgitation, and therefore for the first time in 20 years she was able to have meals with her friends instead of in the privacy of her own room. She has since gained 35 pounds in weight. Later studies showed the entire stomach beneath the diaphragm and in normal position. The patient has continued leading a normal life. It is now 9 years since her surgery and her present age is 83.

Case 2.—I. L. B., a white woman, age 69. We first saw this patient Nov. 15, 1948. Her chief complaint was difficulty in swallowing, with pain in the lower chest while eating. For several years the patient had known she could not eat normally. She had to take her time in swallowing. For the 2 years preceding admission, she had noted severe pain in the lower mediastinal region, and the chest felt full after eating. She noted she had to belch during meals and also had some cough productive of heavy mucoid sputum after eating. She had noted some dyspnea with fatigue for several years, gradually becoming worse although there had been no weight loss. She had an incidental rectal stricture which had to be dilated periodically.

In the past she had had typhoid fever at the age of 16, an ulcer removed from the colon in 1931, a hemorrhoidectomy many years before, and a hysterectomy in 1932. A right nephrectomy had been performed in 1935. A tonsillectomy and antrotomy had also been performed years previously.

The physical examination was negative. The blood work was normal although the blood sedimentation rate was increased to 35 mm. in 1 hour, corrected for cell volume.

A chest film on Nov. 10, 1948, showed a normal lung, heart, and mediastinum with some calcification in the arch of the aorta. Behind the heart shadow there appeared a cystic

cavity with walls about 1 cm. thick, and a fluid level in this space just above the diaphragm. The lateral film revealed the cystic cavity to be the stomach, or a portion of it, above the diaphragm in the posterior mediastinum.

Esophagograms on Nov. 15, 1948, showed the esophagus to be long and redundant, and the major portion of the stomach to be inverted in the lower mediastinum. A diagnosis of esophageal hiatal hernia was made, probably congenital. Because of her age, the loss of one kidney, and her extreme aversion to surgery, we felt an operation could be deferred until such time as she either had obstruction or began to lose weight. She did fairly well under medical management until April 8, 1950, when she was admitted to the hospital vomiting periodically, with some of the vomitus being black at times. She again refused major surgery.

Fig. 1.

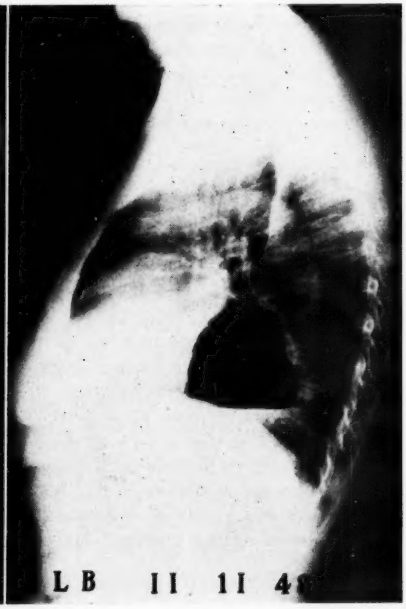

Fig. 2.

Fig. 1.—Case 2, I. L. B. Oct. 20, 1948. A barium swallow demonstrates most of the stomach in the left pleural space with considerable pleural reaction on that side also.

Fig. 2.—Case 2, I. L. B. Nov. 11, 1948. Left lateral chest film. There is a large space behind the heart with an air-fluid level characteristic of a hiatal hernia. The diagnosis can often be made from the lateral film alone.

A new chest film showed pneumonitis in the left mid-lung field. The electrocardiogram showed some left axis deviation. Because of the pneumonitis, surgical repair of the hernia was not insisted upon at the time. Instead, on April 17, 1950, a left phrenic crushing was performed resulting in a good paralysis of the diaphragm. Following that she had some improvement in her ability to swallow but she still had difficulty with foods that contained any coarse vegetable fibers. She was urged to grind all her meat and was instructed to take a teaspoon of Alcaroid after each meal containing meat.

She was next admitted to the King's Daughters' Hospital in Greenville, Miss., Oct. 24, 1950, with what was at first thought to be a heart attack. She was in shock at the time of admission. However, an electrocardiogram was normal except for some left axis deviation. She gave a history of having lost considerable weight in the preceding few

months, although her usual symptoms from the hiatal hernia had been markedly relieved by the phrenic crushing. She was treated medically and gradually recovered.

In 1951, the left diaphragm resumed motion and her symptoms increased. On May 12, 1951, a left permanent phrenic nerve interruption was performed. In December, 1951, she had another attack of retained gastric contents and was treated by Wangensteen suction and intravenous fluids and made a recovery. Fluoroscopic examination at that time showed most of the left diaphragm still paralyzed but there was a suggestion that a few fibers of the nerve were still carrying slight impulses to portions of the diaphragm.

On April 27, 1953, barium studies revealed a large hiatal hernia with at least two thirds of the stomach in the chest. The stomach was markedly dilated with kinking at the diaphragmatic hiatus; it was about half full of fluid. The patient was clinically better at this time, however, than formerly, although she still had occasioal dyspnea and intermittent strangulation of the stomach.

Fig. 3.

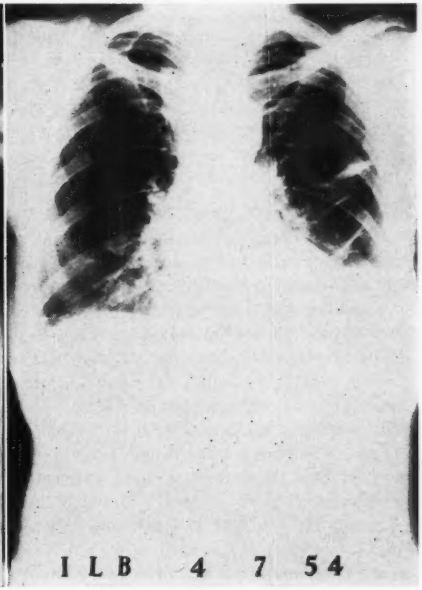

Fig. 4.

Fig. 3.—Case 2, I. L. B. April 8, 1950. The large hiatal hernia shadow merges with the heart shadow and an air-fluid level is seen in the left mid-chest area which is really in the stomach. In addition, there is an aspiration pneumonitis in the left lung. This type of aspiration pneumonitis is common with hiatal hernia and is a very good reason for repairing these hernias before repeated pneumonitis produces permanent pulmonary crippling.

Fig. 4.— Case 2, I. L. B. April 7, 1954. Postoperative film with the stomach now in the abdomen. The left diaphragm is slightly elevated as a phrenic crush was done at the time of surgery to take stress off the suture line. There is some residual pulmonary fibrosis in the left lung particularly from the repeated attacks of aspiration pneumonitis. The patient was clinically well at this time.

In January, 1954, she had a "virus infection" in the lungs with considerable cough and yellowish sputum occasionally streaked with blood. She was placed on a passive bronchial drainage routine, and received antibiotics, with improvement.

On Jan. 12, 1954, she was re-admitted to the Baptist Memorial Hospital with presumably another moderate small meat impaction which responded to medical therapy, including frequent sips of Alcaroid in water, and antispasmodics. The patient wore dentures and

was unable to chew her meat completely. Whenever she ate meat which was not ground, she had spells of impaction or strangulation. On Feb. 7, 1954, she was re-admitted to the Baptist Memorial Hospital with a partially strangulated hiatal hernia. Her clinical condition did not improve and on Feb. 15, 1954, the diaphragmatic hernia was repaired.

At operation a large sliding hiatal hernia was present with part of the stomach and some omentum in the chest. The phrenic nerve was crushed just above the diaphragm. There were rather dense adhesions present between these organs and the hernial sac itself. These were dissected free and the abdominal contents replaced in the abdomen. Most of the sac was excised and part of the sac was used to reinforce the repair behind the esophagus. The diaphragm was slit anteriorly 3 cm. from the esophagus to slide the esophagus forward into a higher position on the diaphragm. The diaphragm was closed behind the esophagus using interrupted sutures of No. 2-0 cotton. The esophageal external wall musculature was then stitched to the diaphragm with interrupted sutures of the same material. Penicillin, 500,000 units, and 1 Gm. of streptomycin were placed in the pleural space and two intercostal tubes were inserted; one in the eighth interspace in the posterior axillary line and the other in the fifth interspace in the anterior axillary line and later connected to a water trap bottle. The incision was closed in layers. The postoperative condition was good and her convalescence was uneventful.

On Aug. 10, 1956, she was hospitalized in the Baptist Memorial Hospital under the care of Dr. Henry Rudner, Sr., with an acute coronary thrombosis from which she slowly recovered. A chest film showed the stomach still in the abdomen beneath the diaphragm. In June, 1957, she reported feeling fine and eating a regular diet without difficulty.

CASE 3.—E. B., a Negro man aged 30, was first admitted to the John Gaston Hospital on Oct. 2, 1943, for presumed intestinal obstruction. At that time, x-ray studies showed a density in the left lower lung field which was located anterolaterally. Barium studies of the upper gastrointestinal tract were normal. A barium enema was not done. He became asymptomatic and was discharged on Oct. 22, 1943.

He was next admitted to the John Gaston Hospital on May 16, 1945, with a history of lower abdominal pain, vomiting, and obstipation of 24 hours' duration, despite administration of oral Epsom salts. The physical examination was negative except for lower abdominal tenderness. Laboratory studies were within the normal range. An exploratory operation was performed on May 15, 1945, through a right paramedian incision with the preoperative diagnosis of small bowel obstruction. At operation a 4 cm. defect was found in the left leaf of the diaphragm, through which the entire greater omentum, transverse colon, and strangulated loops of small intestine had herniated, without a sac. The diaphragmatic opening was enlarged and the hernia reduced. The diaphragm was closed with interrupted silk sutures and the abdominal wound closed in layers.

The patient ran a febrile course and died on May 23, 1945, on the seventh postoperative day. An autopsy revealed massive atelectasis of both lungs.

CASE 4.—J. P., a 28-year-old Negro man, was admitted in shock to the John Gaston Hospital on Aug. 3, 1955, with multiple knife lacerations of the chest wall, right arm, and abdominal wall. Numerous x-ray studies of both chest and abdomen were negative. He was discharged on Aug. 6, 1956.

He was re-admitted on Jan. 10, 1956, with a history of abdominal pain of 6 hours' duration. This was accompanied by nausea and vomiting. Physical examination and laboratory studies were within normal limits. The only significant finding was patchy consolidation in the left base shown in chest films on admission. Subsequent x-ray studies revealed intestinal obstruction and probably a left diaphragmatic hernia.

At operation on Jan. 15, 1956, through a thoracoabdominal incision, utilizing the left sixth intercostal space, the greater omentum was found incarcerated through an opening in the parietal pleura at the eighth left intercostal space in the anterior axillary line. This was due to a diaphragmatic hernia anteriorly at the periphery of the diaphragm. The hernial opening measured 4 cm. in diameter and through it protruded the splenic flexure, the left

half of the transverse colon (which was partially strangulated), and the greater omentum. The hernia was reduced by incising the greater omentum, making it possible to replace the colon in the abdominal cavity. The diaphragm was closed with interrupted silk sutures and the incision closed in layers. Postoperatively the patient became markedly distended to the point that it was necessary to perform a laparotomy Jan. 17, 1956, and cecostomy for decompression. Following this procedure he made an uneventful postoperative recovery and was discharged on Feb. 4, 1956. He was last seen in the outpatient department on March 8, 1956, at which time he was asymptomatic.

CASE 5.—J. G., a 52-year-old Negro man, began to have severe abdominal pain approximately 18 hours prior to his admission to the John Gaston Hospital on March 10, 1947. The pain, which began in the right lower quadrant, soon became localized in the left upper quadrant. There was vomiting numerous times following the onset of the pain. There was no history of previous trauma either to the abdomen or chest. Physical examination revealed a temperature of 98.2° F., respiratory rate of 28, and a blood pressure of 60/50 mm. Hg. He was obviously in profound shock, apprehensive, and perspiring profusely. Significant findings were a tachycardia, abdominal tenderness mostly in the left upper quadrant, and a few audible peristaltic sounds. Laboratory studies showed a red cell count of 5,040,000 and a white cell count of 14,600 with a shift to the left. The urinalysis was not remarkable except for a proteinuria of 2 plus, the blood NPN was 56 mg. per cent on admission.

On x-ray studies of the chest, the diaphragm on the left could not be identified. The colon was not displaced. Films made after the passage of a nasogastric tube indicated rotation of the stomach upward into the left hemithorax. The intestinal pattern suggested that a portion of small intestine was present in the left chest. A diagnosis of strangulated left diaphragmatic hernia was made.

The patient was operated upon on March 10, 1947, through a left thoracoabdominal incision through the eighth intercostal space. Ninety centimeters of gangrenous small intestine were removed from the left chest and resected, and an end-to-end anastomosis performed. The diaphragmatic opening in the central portion of the left leaf was closed with interrupted sutures. The incision was closed in layers with closed intercostal tube drainage employed in the left hemithorax.

Postoperatively the patient had a very stormy course with high fever. On the seventh postoperative day he eviscerated. The evisceration was reduced and closure accomplished with through-and-through sutures, but he died on March 18, 1947, shortly after the procedure. An autopsy was not permitted.

CASE 6.—F. O., a 31-year-old male Mexican, was admitted to the John Gaston Hospital, Oct. 7, 1947, with a history of epigastric pain, nausea, and vomiting of 3 days' duration. The only significant physical finding was left upper quadrant and epigastric tenderness.

Laboratory studies showed a leukocytosis with a shift to the left. X-ray studies of the chest showed the left diaphragmatic outline to be indistinct and an air-fluid level present in the lower left base. A film of the abdomen revealed marked dilatation of the transverse colon.

With a diagnosis of strangulated or incarcerated left diaphragmatic hernia, an exploratory operation was made on Oct. 8, 1947, through a left rectus incision which was extended into a thoracoabdominal incision through the eighth left intercostal space. Through a 2.5 cm. opening posterolaterally in the diaphragm, 25 cm. of transverse colon had herniated and had become strangulated. The hernia was reduced and the diaphragmatic opening closed with interrupted cotton sutures. The incision was closed in layers. His postoperative course was uneventful and he was discharged Nov. 3, 1947.

CASE 7.—W. D., a 50-year-old Negro man, was first admitted to the John Gaston Hospital Aug. 11, 1946, with a pistol bullet wound of the left chest. He ran a febrile course until intercostal drainage was established, with evacuation of a left empyema cavity. Following this procedure his recovery was rapid and uneventful.

He was next admitted on Jan. 15, 1955, with a history of pain in the chest, vomiting, and constipation of 4 days' duration. On physical examination there was limited chest expansion bilaterally. There was a palpable friction rub over the left lateral chest wall, and breath sounds were absent over this area. The abdomen was markedly distended, tympanitic, and silent. Laboratory studies revealed a hemoconcentration, leukocytosis, and an elevated blood NPN (140 mg. per cent). A chest roentgenogram showed a small intestinal obstruction, and an elevation of the left leaf of the diaphragm with atelectasis at the left base.

An exploratory operation was performed on Jan. 16, 1955, through a left thoracoabdominal incision, on a preoperative diagnosis of strangulated diaphragmatic hernia. At operation, 20 cm. proximal to the ileocecal valve, a 25 cm. segment of terminal ileum was found to be strangulated through a 4 or 5 cm. opening slightly posterior to the dome of the diaphragm. This segment, which was necrotic and liquefied, was resected and an end-to-end anastomosis of the terminal ileum performed. During the procedure the patient had a cardiac arrest which responded to cardiac massage and the procedure was quickly terminated. Postoperatively the patient did not breathe spontaneously. He died 40 hours following completion of the procedure on Jan. 18, 1955. Autopsy was obtained and confirmed the above findings.

CASE 8.—R. S., a 34-year-old Negro man, was first admitted to the John Gaston Hospital, Nov. 4, 1945, with a stab wound of the left anterolateral chest wall in the sixth intercostal space. He was observed for possible cardiac stab wound and treated with bed rest until his discharge Nov. 23, 1945. Numerous x-ray and fluoroscopic examinations were reported to be negative.

He was re-admitted to the hospital on Oct. 30, 1951, with an unobtainable blood pressure and a feeble pulse with a rate of 100. He had begun to vomit approximately 36 hours prior to admission and stated that his vomitus was coffee-ground in color. Other findings were absence of breath sounds over the left lower chest and a soft abdomen. Laboratory studies showed a marked hemoconcentration, with an hematocrit of 63 per cent, and a red cell count of 7,800,000. The urinalysis showed a three plus pyuria and proteinuria. Fluoroscopy and barium studies of the gastrointestinal tract revealed an incarcerated diaphragmatic hernia.

At surgery, on Oct. 31, 1951, a left thoracoabdominal incision through the eighth intercostal space was employed. The distal 80 per cent of the stomach along with the entire greater omentum had herniated through a defect in the left diaphragm and had become strangulated. No hernial sac was present. The stomach and omentum were replaced in the abdomen. The diaphragm was closed with interrupted cotton sutures and the incision closed in layers. Closed intercostal drainage was employed in the left pleural space. Postoperatively, the patient became anuric with rising blood NPN to 370 mg. per cent at the time of his death on Nov. 3, 1951. Autopsy was not granted.

CASE 9.—T. L. R., a 36-year-old white man, was admitted to the Baptist Memorial Hospital on Sept. 19, 1956, because of colicky epigastric pain present for 12 hours prior to admission. He stated that he had been stabbed with a pair of short nail scissors over the left chest about 2 years previously. He had been a patient in the Baptist Memorial Hospital at that time but went home 48 hours after admission with no treatment other than observation. In July, 1956, he was hospitalized in the Methodist Hospital, Memphis, Tenn., for epigastric pain and remained in the hospital 2 or 3 days and medication was administered by hypodermic for the pain. At this time he had no cough, sputum, or hemoptysis, but he had noted slight dyspnea on exertion ever since the stabbing occurred 2 years ago. He had to take paregoric occasionally for his nervous stomach. There had been a 20-pound weight loss over the 4-month period prior to admission. The remainder of his history was not related to the present illness.

The physical examination was essentially normal. He was a well-developed, well-nourished white man appearing in good condition with no acute distress. The abdomen showed no mass or tenderness. The liver, spleen, and kidneys were not palpable. An admission chest film here showed a thin-walled cyst or perhaps colon just above the left diaphragm anteriorly. The admission diagnosis was a possible traumatic diaphragmatic hernia with either bowel, stomach, or omentum in the hernia. Lung cyst and tumor were to be ruled out.

A diagnostic pneumoperitoneum was induced and appeared to show a solid organ below and above the left diaphragm apparently going through a hole in the diaphragm anteriorly. Fluoroscopy gave little additional information except for some restriction of motion of the left diaphragm and the finding of this solid density above the left diaphragm, approximately 8 cm. in diameter.

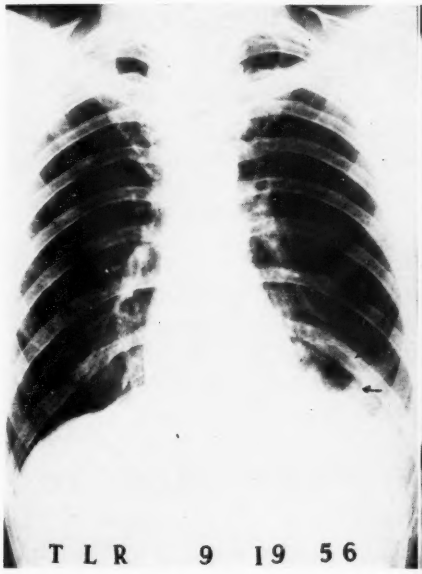

Fig. 5.

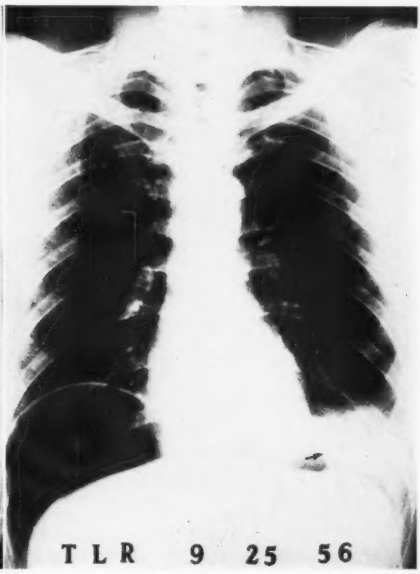

Fig. 6.

Fig. 5.—Case 9, T. L. R. Sept. 19, 1956. A PA chest film shows a poorly defined cystic shadow just above the left diaphragm medially. This abnormality was missed in one or two other hospitals but on hindsight is fairly obvious.

Fig. 6.—Case 9, T. L. R. Sept. 25, 1956. A diagnostic pneumoperitoneum has been induced. There appear to be adhesions between the left diaphragm and subphrenic organs and the stomach shadow is somewhat unusual.

Intravenous pyelograms were normal. An upper gastrointestinal series was done and showed no stomach or colon above the diaphragm.

On Oct. 1, 1956, the left chest was explored through the eighth interspace and a traumatic diaphragmatic hernia discovered with a large mass of omentum above the diaphragm. This omentum was incarcerated and strangulated. There was a traumatic hernial opening anteriorly in the left leaf of the diaphragm about 3 cm. from its anterior insertion into the chest wall. The lung itself appeared normal. The hernial opening measured about 3 cm. in diameter. The omentum was everywhere adherent to the opening and was also adherent to the lower lobe of the lung in several places. These adhesions were ligated and divided. The hernial ring was freed of its adhesions and the omentum was then replaced in the abdomen. However, it was necessary to increase the opening in the diaphragm another

2 cm. in order to reduce the herniated omentum. A two-layer closure was then made of the diaphragmatic opening, imbricating the two edges of the diaphragm, using interrupted mattress sutures of No. 1-0 cotton for the repair. The phrenic nerve was crushed lightly just above the diaphragm. The wound was closed in layers, with one intercostal tube for drainage.

The patient's recovery following surgery was uneventful and he was discharged on Oct. 11, 1956, to resume gradually his former activities.

Fig. 7.

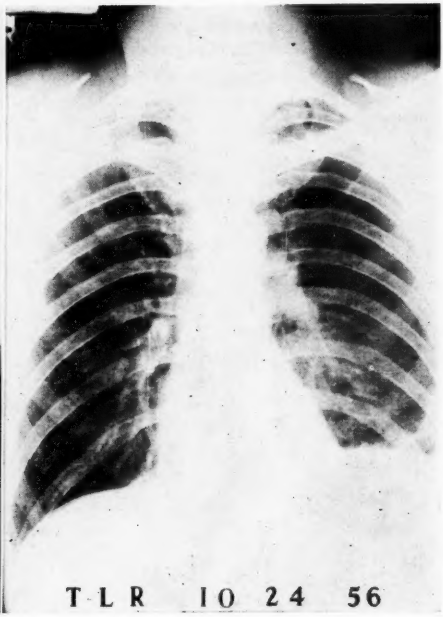

Fig. 8.

Fig. 7.—Case 9, T. L. R. Sept. 26, 1956. This film was taken with the patient lying on his right side. The pneumoperitoneum outlines a solid mass partially above and below the diaphragm which later was proved to be omentum going up through a rent in the diaphragm.

Fig. 8.—Case 9, T. L. R. Oct. 24, 1956. A postoperative film. There is slight pleural reaction on the left which is subsiding and the left diaphragm is slightly elevated due to the phrenic crush which was performed at the time of surgery in order to take stress off the suture line until it healed.

Case 10.—W. P. L., a 52-year-old white man, was admitted to the Baptist Memorial Hospital on April 19, 1951, on the service of Dr. R. L. Sanders and Dr. Turley Farrar. The patient stated that he had had a diaphragmatic hernia for sometime, but 24 hours before admission he had begun to have severe pain in the chest, mostly in the midline anteriorly. He had formerly had a diagnosis also of peptic ulcer. The pain did not radiate and was chiefly substernal in location. Five years before in Birmingham, Ala., a diaphragmatic hernia had been diagnosed. Over the 5-year period prior to admission here he had intermittent spells of vomiting, even on an ulcer diet.

Physical examination revealed a well-developed, well-nourished white man, not appearing acutely ill except for mild tachycardia. The blood pressure was 100/60 mm. Hg and the abdomen showed slight epigastric tenderness but no mass was palpated. The solid organs were not palpable.

Routine laboratory studies were essentially normal. On April 20, 1951, Dr. R. L. Sanders performed an exploratory operation on the patient's abdomen after first having Dr. Farrar perform a left phrenic crush through a neck approach under Novocain anesthesia. Fascia was removed from the left thigh and two sutures were prepared, only one of which was actually needed. When the abdomen was opened it was discovered that about 60 per cent of the stomach was up in the chest and strangulated, with a very large opening in the diaphragm—large enough to admit the entire hand. The stomach was dragged down out of the thorax and the peritoneum freed in front of the esophagus which was then liberated and mobilized. Then the leaves of the diaphragm were elevated with Allis forceps and the opening of about 10 cm. in the diaphragm was closed, using interrupted silk and then continuous fascial sutures. This closed the opening fairly snugly around the esophagus, leaving enough room to admit two fingers beside the esophagus. The stomach and duodenum were entirely negative with no evidence of peptic ulcer. The patient's convalescence was uneventful following surgery and he was discharged on May 5, 1951.

SUMMARY

Case reports are presented of 10 consecutive patients having strangulated diaphragmatic hernias observed in the John Gaston Hospital and Baptist Memorial Hospital, Memphis, Tenn., over a 10-year period. Four of the 10 patients died following surgery, a mortality rate of 40 per cent in this small series.

Strangulated diaphragmatic hernia is a serious surgical emergency with a relatively high mortality. It is suggested that a more aggressive attitude be taken in treating this type of hernia before it becomes strangulated, as surgical repair of the hernia itself is simple with a very low mortality rate when no strangulation is present.

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THE REPAIR OF THORACIC WALL DEFECTS WITH SLIDING RIB GRAFTS

An Experimental Study

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THE most logical source of material to replace defects in the thoracic wall created during the course of major tumor surgery is the patient's own tissue. Available structures, however, have had the disadvantage of instability so that the surgeon is often tempted to add a foreign material to compensate for this.

Faced with a major chest wall defect at the close of a major surgical case, it is the rare instance where the surgeon finds the latissimus dorsi or pectoralis major muscles with their intact neurovascular supply available for repair. Fascia lata or the anterior sheath of the rectus abdominis muscle, tissues most commonly employed for such repair, can be used in most instances. The additional time consumed in obtaining them, or the necessity of having a second surgical team do this, has been their main disadvantage. Furthermore, these tissues do not offer ideal stability in many cases. This disadvantage is more of psychologic than physiologic importance. Closure of the defect simply with the overlying skin results in maximum paradoxical motion in this area. Although this does not significantly impair respiratory exchange, it is unsightly and disturbing to the patient. On occasion, necrosis of such overlying skin has resulted in near fatal pneumothorax.

A variety of heterologus prostheses have been used with the emergence of several basic conclusions. If it is to succeed, the heterologous prosthesis must not only be inert but must also offer both mobility as well as stability. Tantalum plates, used in the past, have been removed almost without exception because they became dislodged or became the focus of an infected sinus.[1, 2, 3] Although valuable in repairing defects that can be covered by muscle and full-thickness skin, tantalum mesh when used as a subcutaneous prosthesis becomes fragmented with slivers of wire protruding from the overlying skin and giving rise to chronic abscess formation.[4, 5, 6]

With this knowledge, the present authors conducted an experimental and clinical study of using sheets of Ivalon sponge as the prosthetic material.[7, 8] Because the vacuoles in this inert material intercommunicate freely, serum

From the Laboratories of Surgical Research, Presbyterian Hospital, University of Illinois, School of Medicine, Chicago, Ill.
Received for publication Jan. 17, 1958.

quickly permeated throughout to lay the framework for the fibroblasts and capillaries which followed. This resulted in a firm yet mobile support of inert substance permeated by living tissue. As in all instances where foreign substances are used, the contraindication to Ivalon being used in the presence of infection, or potential infection, significantly limited its use.

Our experience gave impetus to a further search for a prosthesis which presumably would be autogenous, easily available, offer stability, and yet be mobile. It was felt that a step toward such an achievement would be the development of a technique of providing sliding rib grafts. The ribs adjacent to a thoracic defect are readily available and easily mobilized in a short time and with their intact periosteum can serve as fresh autogenous grafts.

For the sake of greater technical ease, large mongrel dogs were selected for study. Surgery was performed under intravenous Nembutal anesthesia and endotracheal intubation. By removing a 4 cm. length of three consecutive ribs together with the intercostal muscles there was created a full-thickness defect of the right thoracic cage (Fig. 1).

The defect was now ready for repair. The intercostal muscles were separated from the superior (cephalad) aspect of the ribs which had been partially resected (Fig. 1, *A*). This was done lateral to the defect and for a distance sufficient to free an adequate length of rib which was to be mobilized. So that there would be a minimum of bleeding and in order that the neurovascular supply of the intercostal muscle would continue functioning, the muscle was deliberately sectioned at its insertion rather than at its origin. Care was taken not to remove the periosteum.

With a Stryker oscillating saw, using the narrow side of a fan-tail blade, the rib was split lengthwise as far laterally as the intercostal muscle had been freed (Fig. 1, *B*). With a short cross cut the appropriate length of rib was removed. A notch was made on the sectioned ribs medial to the defect so that they could receive the sliding graft. With a very small drill (preferably a dental drill) holes were made at the ends of the grafts and of the sectioned ribs on either side of the defect.

No. 32 stainless steel wire was passed through these holes and, with a figure-of-eight tie, the sliding grafts were secured in position. By trial and error the above ligature and tying techniques were found to be the most suitable to secure the graft and to prevent ligature fragmentation. After all the rib grafts were secured, the intercostal muscle was re-attached to the corresponding rib (Fig. 1, *C*). Because half the width of the rib was absent, the intercostal muscle had to be stretched somewhat in order to reach the rib below. Of course, over the site of the original chest wall defect, sliding rib grafts but no intercostal muscles were present. The overlying musculature and skin were then closed in layers.

At the conclusion of the surgery the rib grafts by their solid support prevented paradoxical motion of the thoracic wall, and yet were mobile in concert with the remaining ribs. In effect these were immediate autogenous rib grafts with intact periosteum. The majority (90 per cent) of the grafts survived and developed into viable solid supporting structures. In no instance did infection develop. The rib grafts that did not survive were absorbed. By the time this

was evident, fibrous tissue had sufficiently infiltrated this area so that the defect could be detected only by x-ray. Occasionally there developed a pseudoarthrosis at the point where the sliding graft was anchored. This could be demonstrated by x-ray (Fig. 2) but was not detrimental.

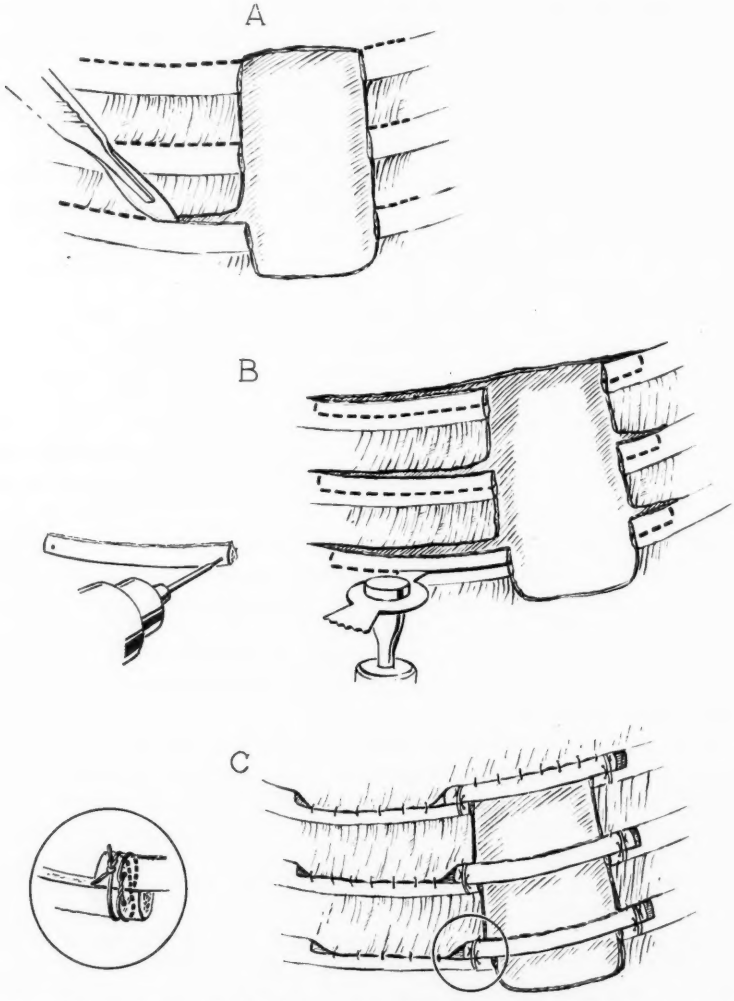

Fig. 1.—Composite drawing of operative technique of sliding rib grafts.

A, Intercostal muscle is divided at its insertion and sufficiently laterally to allow an appropriate length of rib to be mobilized.

B, The cephalad portion of the rib with its intact periosteum is split lengthwise with a Stryker oscillating saw. A short notch is made in the ribs medial to the defect. Holes are drilled at all the rib ends.

C, The grafts are anchored in place with figure-of-eight ties, using No. 32 stainless steel wire (see insert). The intercostal muscles are then re-attached to the corresponding ribs.

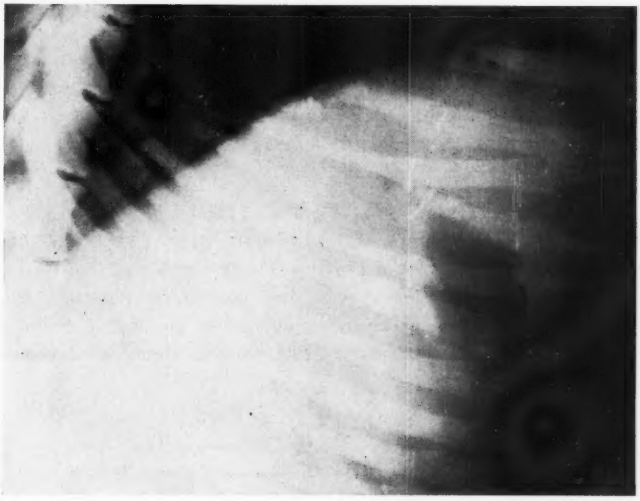

Fig. 2.—Lateral chest x-ray in a dog having sliding rib grafts performed 20 weeks previously. One graft did not "take" and pseudoarthrosis is present in two other areas. Neither of these deficiencies resulted in functional impairment.

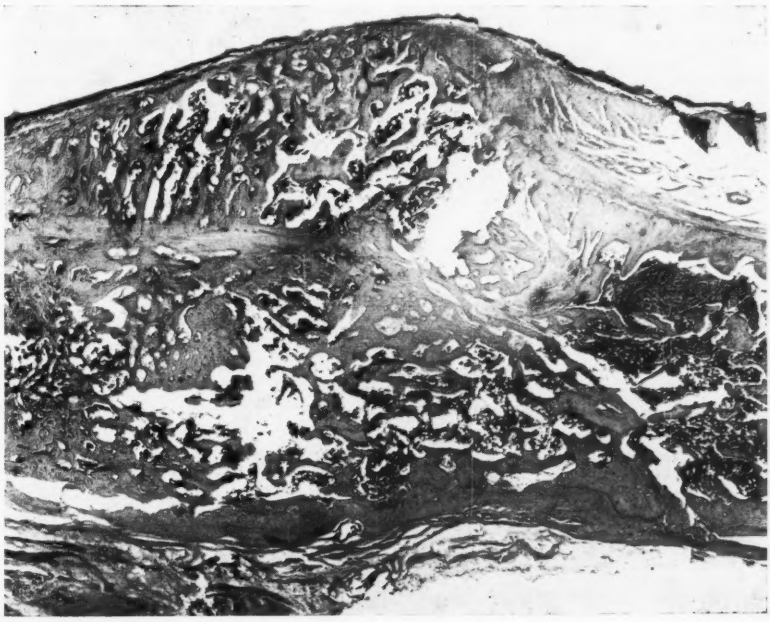

Fig. 3.—This is a photomicrograph of a sliding rib graft junction at the end of 20 weeks. In the upper right portion of the picture is the sliding rib graft. It is indistinguishable from the ribs (left lower portion of picture) to which it has been anchored.

The procedure was performed on 15 animals; some on both sides. The animals were sacrificed at intervals of 2 to 50 weeks. At autopsy, there were variable lung adhesions to the surgical site but most of the area was covered with parietal pleura. The grafted region was as firm if not more so than the adjacent tissues. On microscopic examination the grafted ribs are indistinguishable from those on the recipient site (Fig. 3).

SUMMARY

In the process of excising chest wall tumors, or when, because of locally invasive bronchogenic carcinoma, a portion of the chest wall has to be sacrificed along with the lung, there is the necessity of adequately repairing the resultant defect. To meet this problem we have developed in dogs a technique of immediate autogenous sliding rib grafts. The high incidence of success in animals justifies clinical application.

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THE ARTERIAL DISTRIBUTION TO THE RIGHT UPPER PULMONARY LOBE

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WE HAVE previously reported[1] on the arterial distribution to the left upper lobe, a subject on which there has been considerable misinformation in the literature. While collecting data for the above report, we have used the same type data sheets to accumulate information on the arterial distribution to the right upper lobe. Although the arterial supply to the latter is considerably more constant, we have encountered 13 variations in 200 cases. It is felt that a set of simple "maps" may be useful to residents in training and to the occasional operator.

METHOD OF STUDY

Each right upper lobectomy or segmental resection of the right upper lobe performed at Herman Kiefer Hospital was charted immediately after surgery as to the distribution of the arteries. As a teaching procedure for the resident staff, each arterial stem was traced to its first major branching and the destination of these branches determined. Those segmental operations were excluded in which calcification and scarring would have rendered hazardous dissection of vessels other than those which required ligation for the proposed operation. No attempt was made to subdivide the individual pulmonary segments or the arteries thereto. While the identification of the secondary and tertiary branches of the segmental arteries is a delight to the anatomist, it has relatively little surgical importance. The present report is based on 200 cases where the entire arterial supply to the right upper lobe could be clearly identified and traced.

RESULTS OF THE STUDY

From Table I it is evident that in nearly three quarters of cases the right upper lobe is supplied by two arterial trunks.

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Received for publication Jan. 20, 1958.

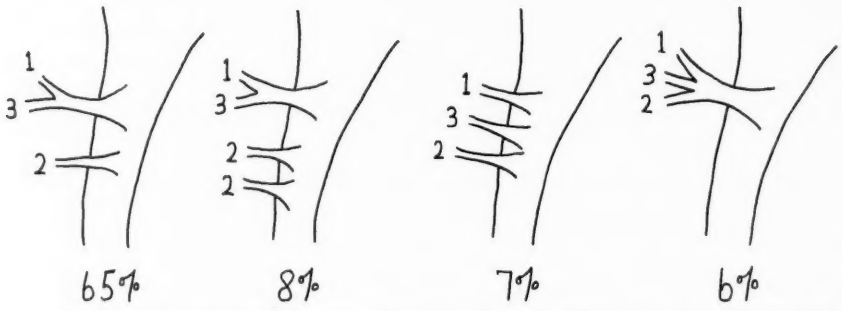

Fig. 1.—The most common arterial distributions to the right upper lobe. These four patterns account for 86 per cent of all cases. In all figures, the Jackson-Huber classification is used with *1* representing the apical, *2* the posterior, and *3* the anterior segmental arteries.

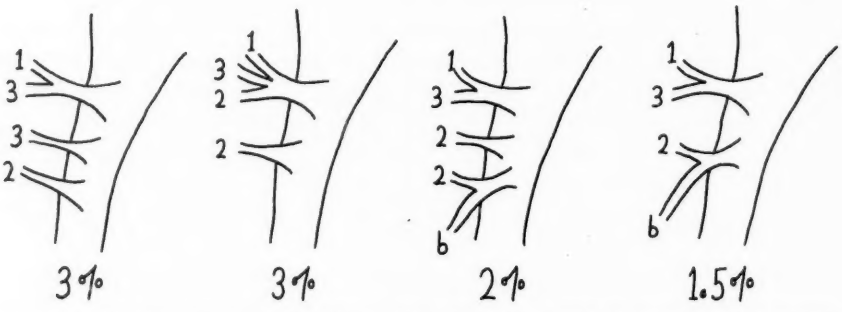

Fig. 2.—Less common distributions. Note the 3.5 per cent of cases in which supply to the posterior segment is derived from the artery to the superior division of the lower lobe.

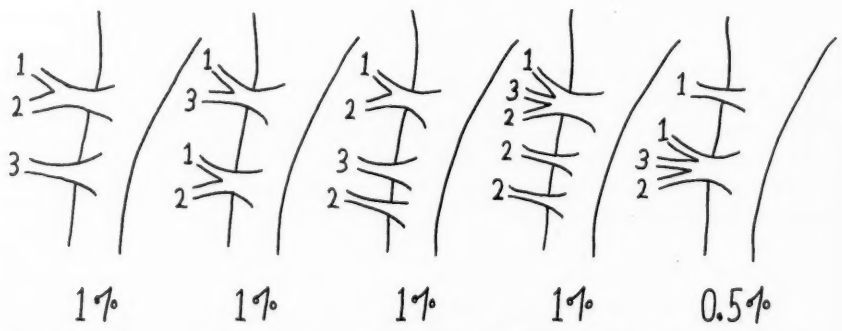

Fig. 3.—Least common distributions. These five variations are found in only 4.5 per cent of cases.

TABLE I. NUMBER OF ARTERIAL TRUNKS TO THE RIGHT UPPER LOBE

NUMBER OF BRANCHES	PER CENT OF CASES
1	6
2	72
3	22
Total	100
Mean 2.2	

The illustrations are more or less self-explanatory. In Fig. 1, it is seen that 65 per cent of all cases have an apico-anterior and a posterior trunk. In another 8 per cent, there are two posterior trunks. The four distributions shown in Fig. 1 account for 86 per cent of all cases. Fig. 2 reveals that, in 3.5 per cent of cases, all or part of the arterial supply to the posterior segment will be derived from the artery to the superior segment of the lower lobe. In the 7 cases in which this occurred, the branching was so proximal that it was immediately recognized and the supply to the superior division was saved in all instances. Fig. 3 shows the least common form of distribution, occurring in less than 5 per cent of cases.

Table II shows the number of individual branches supplying each segment. It will be noted that, in the great majority of cases, each segment is supplied by only one artery.

TABLE II. NUMBER OF INDIVIDUAL BRANCHES TO EACH SEGMENT

	SEGMENT 1	SEGMENT 2	SEGMENT 3
One artery	98.5	85	97
Two arteries	1.5	14	3
Three arteries	0.0	1	0
	100%	100%	100%

CONCLUSIONS AND SUMMARY

Two hundred cases are presented in which the arterial distribution to the right upper lobe has been accurately mapped. No startling anomalies have been encountered. Three quarters of all cases are supplied by an apico-anterior and either one or two posterior trunks. In the remaining quarter of the cases, eleven additional distributions have been identified. In the latter cases perhaps the most significant are the 3.5 per cent where the posterior segment is supplied from the artery to the superior division of the lower lobe.

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EVENTRATION OF THE DIAPHRAGM: HERNIATION OF THE LIVER INTO THE LEFT AND RIGHT HEMITHORAX

A Case Report

Moses Grossman, M.D., Rita Brady, M.D., and H. Brodie Stephens, M.D., San Francisco, Calif.

THIS is a report of a case of eventration of the diaphragm occurring in a 5½-year-old boy. The only abdominal organ extending into the chest was the liver, which was found in the right as well as the left chest.

There is extensive reporting on the subject of eventration of the diaphragm with herniation of the liver into the chest, otherwise known as partial eventration of right diaphragm, superior accessory lobe of liver,[1] or hernia diaphragmatica dextra hepatica.[2] Several excellent reviews of the literature on this subject have appeared recently.[3-6] The majority of reported cases have been those of right-sided herniation. A careful review of the literature reveals no previously reported cases of bilateral eventration of the diaphragm. We would like to report such a case in detail, one which simulated an intrathoracic tumor.

CASE REPORT

D. H., a 5½-year-old boy, was referred from Anchorage, Alaska, with the chief complaint of deformity of chest, poor growth, and a "mass in the chest." The child was born prematurely, weighing 2.64 kilograms (3 pounds 10 ounces), following a normal pregnancy and delivery. Both the father (39 years old) and the mother (42 years old) were in good health. Family history revealed a half sister, 19 years old, who had had a neurofibroma about the size of a walnut removed from the mediastinum. Her recovery was uncomplicated.

The first 5 years of life were uneventful except for the occurrence of whooping cough at the age of 4 months. The first chest film was made approximately 8 months before admission here because of a prominence of the cartilages over the left chest, and revealed a mass within the chest confluent with the cardiac shadow. The child was completely asymptomatic as far as the heart and lungs were concerned.

Physical examination at the time of admission revealed a very slender, asthenic boy who appeared quite lively and sociable, although his over-all performance gave one the impression of mild retardation. He weighed 13.62 kilograms (30 pounds) and was 82 cm. tall. His head circumference was 46 cm., which is in the 50 percentile for a 9-month-old child.

The positive physical findings other than his small size were limited to the chest. There was a mild, left precordial bulge with prominent junctions of the cartilages and ribs. No abnormal heaves or thrills were noted. Careful percussion revealed an increased

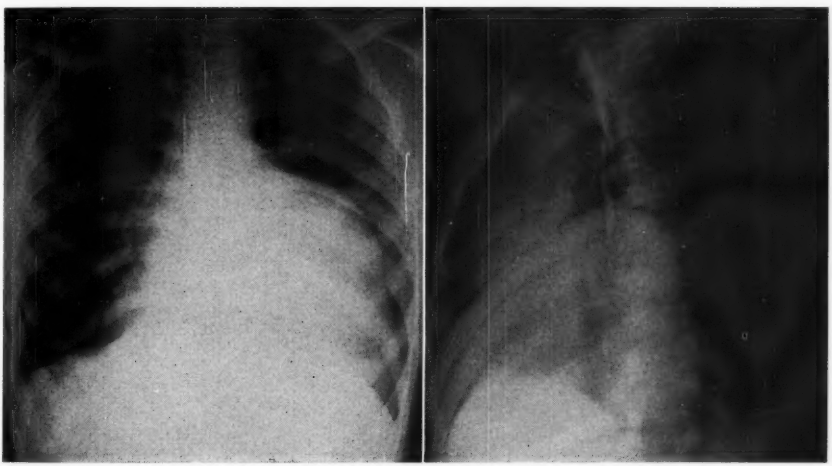
From the University of California Medical Center, San Francisco 22, Calif.
Received for publication Jan. 27, 1958.

area of dullness in the lower mediastinum. The breath sounds were of good quality throughout. The heart rhythm was regular, the rate was not remarkable, the heart sounds were of good quality and the second sound was single and of normal intensity. A Grade 2 systolic murmur was best heard at the apex; this changed with position and had the quality of a functional murmur. The pulses were of good quality throughout. The blood pressure was 112/75 mm. Hg.

Laboratory work revealed a hemoglobin of 12.3 Gm., packed cell volume 40, white cell count 10,150, normal differential; urine clear; tuberculin 1:1000 negative, Schick negative; Echinococcus skin test, negative. A psychometric examination revealed his mental age to be 4 years 4 months, with a borderline classification of intelligence.

An electrocardiogram showed a notched, slurred S in Lead V_1 which could indicate an impaired right conduction but also could well be within normal limits.

Roentgenograms of the chest revealed a large homogeneous mass in the lower anterior portion of the thorax which was contiguous with the contour of the anteromedian segment of both the right and left hemidiaphragm as well as the base of the cardiac silhouette (Fig. 1).



A. *B.*

Fig. 1.—*A*, Roentgenogram of chest in the posteroanterior projection. Note large homogeneous mass contiguous with the base of the cardiac silhouette which is rotated and displaced cephalad.

B, Left anterior oblique projection of chest demonstrating contiguity of the mass to the cardiac shadow and both leaves of the diaphragm.

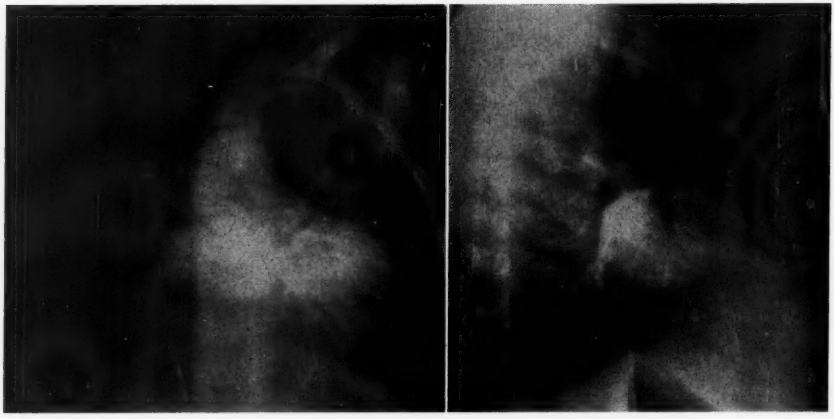
Fluoroscopic examination of the chest disclosed no abnormality of the diaphragmatic excursion. The mass which moved synchronously with both leaves of the diaphragm exhibited no alteration in size or shape with respiration and no intrinsic pulsation was detected. An angiocardiogram revealed no intrinsic abnormality of the cardiac chambers or of their sequence of opacification. The cardiac silhouette, however, was compressed and displaced cephalad and slightly posterior by the large mass, in which no contrast material was detected (Fig. 2). Although the study did not conclusively establish the location of the mass, a tentative diagnosis of a mediastinal tumor—most probably a pericardial cyst—was made.

A bilateral exploratory thoracotomy and a limited laparotomy were performed on March 29, 1957 (H. B. S.). A transverse incision was made, centered over the fourth interspace, and carried from one anterior axillary line to the other and across the sternum. Both pleural cavities were entered. Later, in order to more clearly define the abnormality,

the inferior segment of the sternum was divided in the midline and a "T" extension of the incision carried through the anterior abdominal wall into the peritoneal cavity. This latter incision extended downward almost to the umbilicus.

A thorough exploration of the chest was performed, as well as a limited exploration of the peritoneal cavity. The findings appeared to be a failure of development of the tendinous portion of both hemidiaphragms. The defective development involved the central tendon of the diaphragm only. The muscular portion appeared entirely normal. Both the pleural and the peritoneal cavities were intact.

The liver protruded through this central portion of the diaphragm and presented itself in the chest just posterior to the lower segment of the sternum. It was located in the midline just beneath the heart and had a bilobular appearance, as illustrated in Fig. 3. The left lobe of the liver was abnormally enlarged. The round ligament seemed to be in the normal location and the right lobe of the liver was thought to be of normal size. No attempt was made to determine the location of the gall bladder. A biopsy was taken from the left lobe of the liver and revealed entirely normal liver tissue. It was not thought possible to repair the very large defect which involved the central portion of the diaphragm and the abdominal and chest incisions were closed. The child had an uneventful hospital course and was discharged on the twelfth postoperative day.



A. *B.*

Fig 2.—*A* and *B*, Angiocardiogram in anteroposterior and left lateral projection showing displacement and rotation of heart by nonopacified mass.

DISCUSSION

The roentgenographic findings associated with partial eventration of the right leaf of the diaphragm with herniation of the liver are usually sufficiently characteristic[3-5] so that the diagnosis can be reached with ease. However, in this case, the location of the mass in the chest immediately inferior to the heart and centrally in the chest was misleading. To our knowledge, eventration of the diaphragm in this particular location has not been reported previously. The angiocardiogram was performed to further ascertain the relation of the anteromedian mass to the heart and to the diaphragm. This clearly revealed that the mass was extracardiac, but did not clarify its position with respect to the diaphragm.

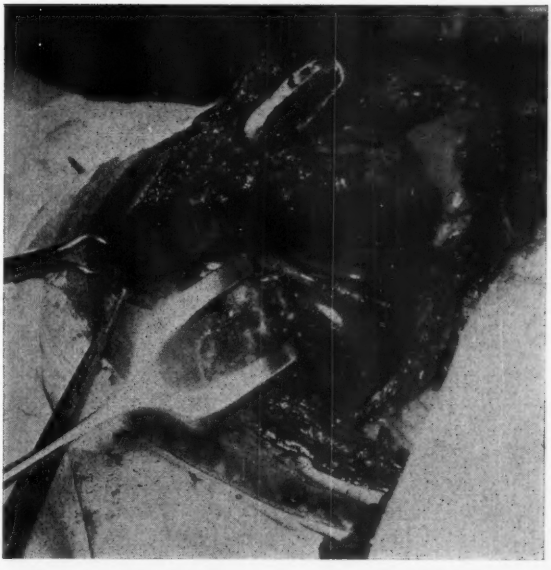


Fig. 3*A*.—Surgical Exposure. The patient is in the supine position. The head is to right and the feet are to left.

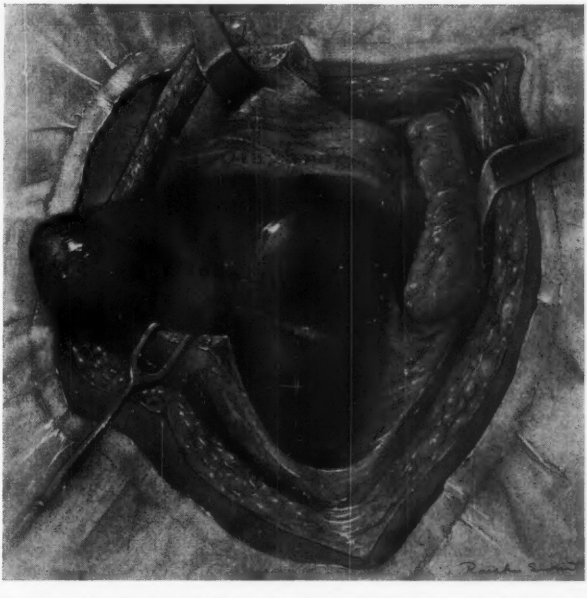


Fig. 3*B*.—Note protrusion of enlarged left lobe of liver into the central portion of the thorax.

In retrospect it is apparent that a diagnostic pneumoperitoneum would have been the roentgenographic procedure of choice and would have led to the proper diagnosis preoperatively.[7] This is illustrated by Fig. 4 which depicts another case in which there was a partial eventration of the left leaf of the diaphragm with herniation of an abdominal viscus.

The more commonly described cases of eventration of the right diaphragm and herniation of the liver into the right chest are due embryologically to a failure of formation of the muscular portion of the diaphragm.[3, 4] In the case reported here, the developmental failure responsible for the eventration was

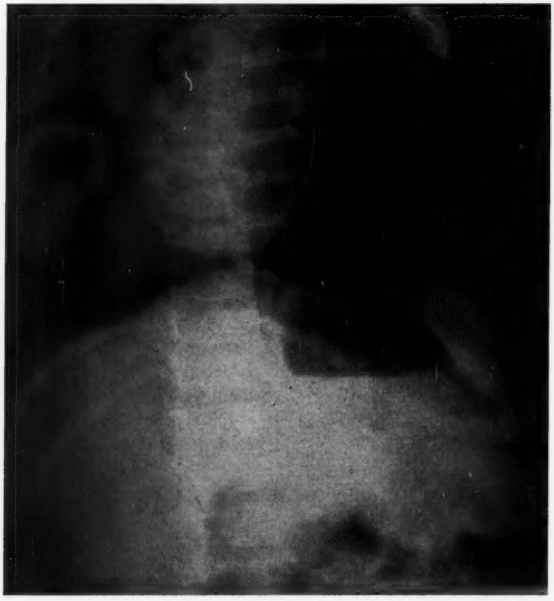


Fig. 4.—Film of abdomen following diagnostic pneumoperitoneum demonstrating intact diaphragm.

failure of formation of the central tendon which derives from the central portion of the septum transversum. The muscular part of the diaphragm appeared normal but, instead of a strong firm central tendon, only the thin pleural and peritoneal layers were present.

This, therefore, represented a congenital malformation with nondevelopment of the central tendons of the diaphragm. The physiologic intra-abdominal pressure stretched the very thin membranous central portion of the diaphragm, enabling a portion of the liver—involving both lobes—to prolapse into the diaphragmatic pouch thus developed.

The importance of recognizing eventration of the diaphragm with herniation of the liver preoperatively has been stressed previously.[4, 7, 8] In our case, we were misled by the fact that the eventration occurred in the area of the central

tendon of the diaphragm instead of the mucular portion of the right or left side. This case is being reported to call attention to the possibility of this diagnosis in those cases where the suspected intrathoracic mass is located centrally.

SUMMARY

This is the report of a case of eventration of the diaphragm with herniation of the right and left lobes of the liver into both sides of the chest, due to failure of proper formation of the central portion of the central tendon of the diaphragm.

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Members Deceased

RUDOLPH MATAS
FRANK I. TERRILL
GABRIEL TUCKER

ANNUAL MEETING NOTES

The Thirty-eighth Annual Meeting of The American Association for Thoracic Surgery was held in Boston, Massachusetts, May 16, 17, and 18, 1958. There were 1,157 physicians who registered for the scientific sessions. In addition there were 142 wives present.

The first day of this year's meeting was dedicated to the memory of Evarts A. Graham. It is planned that the papers read on this day will comprise a special issue of THE JOURNAL OF THORACIC SURGERY and be appropriately marked. To further perpetuate his memory the Traveling Fellowship of this Association has been renamed The Evarts A. Graham Memorial Traveling Fellowship. Mrs. Graham was an honored guest of the Association during this meeting.

The headquarters office of the Association has proved itself over the two-year period that it has been in operation. It, therefore, becomes evident that the Association should no longer continue to impose upon the generosity of Washington University for this space. To this end the necessary funds were appropriated with which to set up an administrative office in the St. Louis area. The new address of this office will be Carondelet Building, 7730 Carondelet Avenue, St. Louis 5, Missouri.

The problems posed by the unprecedented growth of the Association and the report of the special committee that has studied them for two years were discussed at great length in Executive Session. However, no clearly acceptable method for strictly limiting the membership was evolved. The size of the attendance at our annual meetings is undergoing further scrutiny.

The following changes in membership status were voted by the Association during its Executive Session:

Seven members were dropped from the rolls of the Association either through the acceptance of their resignations or because of nonattendance.

The following Active members were elevated to Senior status:

Andre Cournand
John H. Garlock
Harlon W. Harrison
Harold A. Kipp
Charles W. Lester
Alton Ochsner
Forrester Raine
Arthur S. W. Touroff

The following Associate members were elevated to Active membership:

Richard H. Adler
Neil C. Andrews
John W. Bell
Ralph Berg, Jr.
Jacques Bruneau
Robert Fox
Sanford W. French III
Frank Glenn
Alfred Goldman
Joffre-Andre Gravel
James D. Hardy
Ormand C. Julian
Winfield O. Kelley
C. Frederick Kittle
Harvey J. Mendelsohn
Bertrand W. Meyer
Andrew G. Morrow
Paul Nemir, Jr.
Melvin M. Newman
Francis J. Phillips
Benson B. Roe
John M. Salyer
Hawley H. Seiler
John Y. Templeton III
Gordon W. Thomas
Harry E. Walkup
Elton Watkins, Jr.

The following candidates were elected to Associate membership:

Jay L. Ankeney, Cleveland, Ohio
Raymond J. Barrett, Detroit, Mich.
Charles A. Beskin, Baton Rouge, La.
William S. Blakemore, Philadelphia, Pa.
Ivan W. Brown, Jr., Durham, N. C.
John C. Callaghan, Edmonton, Alberta, Canada
Gilbert S. Campbell, Minneapolis, Minn.
Francis H. Cole, Memphis, Tenn.
E. Stanley Crawford, Houston, Texas
Julio C. Davila, Philadelphia, Pa.
Gerard Desforges, Melrose, Mass.
Thomas B. Ferguson, St. Louis, Mo.
Charles W. Findlay, Jr., New York, N. Y.
Joseph M. Ford, New York, N. Y.
Raleigh M. Hood, San Leandro, Calif.
Charles A. Hufnagel, Washington, D. C.
Karl E. Karlson, Brooklyn, N. Y.
Harvey W. Kausel, Albany, N. Y.
Jerome Harold Kay, Los Angeles, Calif.
John Lester Kee, Dallas, Texas
James Alastair Key, Toronto, Ontario, Canada
Earle B. Mahoney, Rochester, N. Y.
Arthur C. Miller, Loma Linda, Calif.
Joseph D. Morris, Ann Arbor, Mich.
Thomas F. Nealon, Wynnewood, Pa.
Adrian J. Neerken, Kalamazoo, Mich.
Russell M. Nelson, Salt Lake City, Utah
Rush E. Netterville, Jackson, Miss.
Rex Beach Perkins, Birmingham, Ala.
Morris Rubin, Bronx, N. Y.
David C. Sabiston, Baltimore, Md.
Morris W. Selman, Toledo, Ohio
Frank C. Spencer, Baltimore, Md.
David State, Los Angeles, Calif.
Lawrence H. Strug, New Orleans, La.
Timothy Takaro, Oteen, N. C.
Alfred M. Tocker, Wichita, Kan.
Mark W. Wolcott, Coral Gables, Fla.